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POLIOMYELITIS WITH SPECIAL REFERENCE TO THE DRINKER RESPIRATOR THERAPY*

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EACH year, more and more information concerning acute anterior poliomyelitis is placed on record. Some reports deal with the etiology of the disease, others with the diagnosis and many suggest new forms of therapy.

In the last six months of the year 1937, a fairly large number of cases of poliomyelitis were admitted to the University and Minneapolis General Hospitals. These patients were closely followed and interesting observations were made. The majority of the patients were admitted during the months of August and September. Three-fourths of these were between the ages of one and fourteen and the lowest mortality was found in the years from five to nine. There was an equal distribution as to sex. The size of the family did not seem to influence the incidence of the disease. There were no cases of second attacks.

In this series, the average maximum temperature was 103.9 F. and the white blood cell count averaged 8,900. The leukocyte count showed no definite relation to the temperature, indicating the blood response and presumably the antibody response to be slow or absent. The majority of the spinal fluid cell counts were between 50 and 100. The cell count generally fell gradually from the level found at the onset of the disease until the period just preceding the most extensive paralysis. At this time it rose again, falling slowly thereafter. The polymorphonuclear cells in the spinal fluid slowly decreased as the disease progressed and the mononuclears correspondingly gradually rose until about the fourteenth day, when they were practically one hundred per cent. The course of the disease could not be predicted by the temperature, the white blood cell count or the spinal fluid findings.

The distribution of the paralysis was of interest. There were fourteen cases with paralysis of the muscles of the trunk and abdomen. This was a higher number than was expected. Most outstanding, however, was the incidence of cases having respiratory difficulty. Minnesota has experienced during the past ten years three periods during which there have been an unusual number of cases of poliomyelitis. Each time there has been a corresponding increase in the number of patients admitted to the hospitals. Among the hospital cases the incidence of patients with difficulty in breathing has increased until during the latter half of the past year it reached a rather alarming figure. Seventy-nine cases of poliomyelitis were admitted to the University and Minneapolis General Hospitals from July 1, 1937, to December 31, 1937, and of this number twenty-nine patients had some respiratory distress. Tables I and II summarize these observations.

Thunberg, in 1926, first used the barospirometer for artificial respiration. The patient was entirely enclosed in an air-tight cylinder. Drinker, Shaw and McKhann[4,5] reported, in 1929, a modification of the barospirometer so that the patient's head could be outside the air-tight cylinder. Since then the Drinker respirator has been widely used for the treatment of respiratory failure in poliomyelitis. In Minnesota, however, where respirators are scarce, physicians must

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TABLE I. CASE INCIDENCE OF POLIOMYELITIS DURING THE PAST TEN YEARS

Year	University and General Hospitals	City of Minneapolis	State of Minnesota
1928	14	34	223
1929	14	4	30
1930	13	46	475
1931	65	143	810
1932	8	10	122
1933	47	94	382
1934	9	9	109
1935	6	13	98
1936	6	8	31
1937	83	95	358

TABLE II. TYPES OF POLIOMYELITIS DURING YEARS OF HIGHEST INCIDENCE

University and Minneapolis General Hospitals

TYPE	1931	1933	1937*
Abortive	1	0	6
Non-paralytic	31	17	18
Encephalitic	1	0	1
Paralytic			
Spinal	26	17	25
Spinal respiratory	1	2	8
Bulbar	4	7	15
Bulbo-Spinal	1	4	6

*Six months period (July 1, 1937, to Dec. 31, 1937).

know the indications for this form of therapy especially in view of the fact that the recent small epidemics of poliomyelitis have been accompanied by a high incidence of cases with respiratory embarrassment. Some patients respond very well to the Drinker respirator; others do not respond and are even made worse. Physicians tend to place all their cases with respiratory difficulty into the machine. The authors feel that this is a poor procedure and offer, therefore, in this brief communication, the newer knowledge of the Drinker respirator therapy.

The most important problem is the careful selection of the patients for treatment. The following classification has been found to be of great assistance.

Spinal Cases

Spinal cases in which there is a direct paralysis of the muscles of respiration innervated by the nerves from the dorsal and cervical spinal cord. The intercostal muscles, the diaphragm, or both, are involved. Paralysis of the intercostal muscles is determined by watching the expansion of the chest during inspiration. The magnitude of expansion is more readily estimated by pressing firmly on the upper abdomen, thus diminishing the excursion of the diaphragm. Paralysis of the diaphragm is determined by observing the movement of the abdominal wall during inspiration. If there is no abdominal movement, or if the abdomen is drawn inward during inspiration, it indicates weakness or paralysis of the diaphragm.

The individual with spinal respiratory paralysis usually advances to the stage where he lies in bed motionless, the eyes having a fearful stare. The face is dusky and the lips are cyanotic. The alæ nasi dilate with every inspiration and the accessory muscles of respiration, especially the sternocleidomastoid, are visible with every inspiration. The chest and the abdomen become practically vibrationless. The patient cannot sleep, but when he is placed in the Drinker respirator the response is usually dramatic. The cyanosis disappears and the face assumes its natural color. The facial expression is that of satisfaction. After the patient's breathing and the respirator become synchronous, the individual falls into a deep sleep which may last many hours.

The early respirators had three speeds: 15, 30 and 45 per minute. Later models had two speeds: 15 and 30 per minute. The latest type is so constructed that all degrees of speed can be obtained and the machine can be tilted as desired. Eight of our patients with respiratory difficulty were of the spinal type and all were placed in the Drinker machine. None of these died in the respirator although one died after removal from the machine, of peritonitis which followed a recto-vaginal fistula. The initial rate of 30 per minute was used in the majority of our cases, and then the rate was adjusted to correspond as nearly as possible to the normal respiratory rate of the patient—faster in children and slower in adults. Children were started at about 10 cm. of negative water pressure, which was gradually increased with age to 15 cm. or 20 cm. for adults. In each case the machine was operated at the lowest negative water pressure at which the patient had complete relief of symptoms.

The patients placed in the respirator remained in it undisturbed except for rotation from side to side several times daily. The efficiency of the respiratory muscles was observed when the machine was opened to give nursing care. If

improvement began, the respirator was opened for longer intervals and then, following the suggestions of Emil Smith,[12] the patients were asked to cough. If they could, they were removed from the machine and watched carefully for cyanosis. However, when the patients were able to breathe without effort but could not cough, they were kept a part of each day in the machine until such time as they were able to cough.

There was some mechanical difficulty in giving adequate nursing care to the patients in the respirator. The disadvantages of the machine were as follows:

1. The temperature was difficult to regulate.
2. The frequent incontinence of urine and sometimes of feces increased the tendency to skin irritation.
3. The rubber collar was uncomfortable and irritated the neck.
4. Early orthopedic treatment could not be carried out as desired.

Bulbar Cases

Bulbar cases in which there is involvement of the nuclei of the cranial nerves with apparent injury of the respiratory center. The damage to the bulbar nerves supplying the muscles of the pharnyx and the larynx is often ushered in by a nasal quality of the voice, by choking attack or by regurgitation of fluids through the nose. Various grades of aphonia and impaired ability to cough or swallow are present. Advanced cases may be heard breathing with an ominous gurgling in the throat. Attempting to swallow causes a choking attack. Secretions around the glottis which seem to prevent deep inspirations, cause the breathing to simulate paralysis of the respiratory muscles. Deep inspirations tend to aspirate fluid into the larynx and are frequently interrupted by a forced expiration. However, extreme irregularity of breathing, both as to depth and rhythm, is an indication of damage to the respiratory center.

Nine of our patients had pharyngeal paralysis with some interference in proper breathing. None was treated with the Drinker respirator and all recovered. Two patients also had facial paralysis, and this cleared up satisfactorily. There were, however, six bulbar cases with definite respiratory difficulty. Two patients were placed in the Drinker respirator but they did poorly and expired in a short time. It was apparent that the machine caused aspiration of material from the throat and at the same time overcame the reflex choking and coughing by which this material could be ejected. The next case, therefore, did not receive the respirator therapy, the following measures being substituted:

1. The foot of the patient's bed was elevated to facilitate drainage of secretions away from the larynx and into the posterior pharynx, where they were removed by suction.
2. Postural drainage was used during the choking spells when the condition of the patient permitted.
3. Nothing was given by mouth and vomiting was almost eliminated. Plenty of parenteral fluid was administered and large whole blood transfusions were also given.

In spite of these procedures, the patient rapidly became worse. Choking attacks occurred with temporary severe anoxemia resulting in deep cyanosis. Aspiration of mucus and vomitus into the bronchial tree took place, producing evidence of bronchial obstruction leading to a complete cessation of breathing at times. Excessive fatigue due to the continual interference with inspiration by the unswallowed material in the pharynx or the actual inspiration of this material was the cause of death. However, the three remaining patients did not have such an extensive involvement of the bulbar nerves as was present in the previous case. All were treated without the Drinker respirator by the method outlined above. At first, it was difficult to refuse the use of the respirator to these cases, but as they progressed favorably the thought of using the machine vanished.

Bulbo-spinal Cases

Bulbo-spinal cases have, as the term indicates, both spinal and bulbar involvement. This is manifested by paralysis of intercostal muscles, the diaphragm, or both, and some paralysis of the pharynx. If the paralysis of the pharynx is unilateral, as indicated by difficulty in swallowing, the respirator may be effective. If the paralysis of the pharynx is bilateral, as indicated by a total inability to swallow, the machine is ineffective. This was easily demonstrated by the fact that five of our six bulbo-spinal patients were placed in the Drinker respirator, and none survived. They all had a bilateral paralysis of

TABLE III. REPORTS IN THE LITERATURE OF POLIOMYELITIS CASES TREATED WITH THE DRINKER RESPIRATOR AND THE RESULTS OBTAINED

Year Author	Spinal Respiratory Treated	Spinal Respiratory Died	Pure Bulbar Treated	Pure Bulbar Died	Bulbo Spinal Treated	Bulbo Spinal Died
1929—Drinker & McKhann[4]	1	1				
1930—Shambough, Harrison & Farrell[10]	1	0			2	2
1931—Shaw, Thelander & Limper[11]	10	1	5	5		
1932—Wilson[14,15]	23	3	20	13		
1932—Wesselhoeft & Smith[13]	9	0	5	4	16	13
1933—Harper & Tennant[7]	7	0	17	17		
1934—Crone[3]	9	5	5	3		
1934—Landon[8]	56	29	2	2	30	22
1935—Fischer & Stillerman[6]	8	0	10	7		
1936—Brahdy & Lenarsky[1,2]	51	24	12	12		
1937—Stoesser & Sako	8	0	2	2	5	5
Grand Total of Cases	183	63	78	65	53	42
Mortality Per Cent		34		83		80

the pharynx. The one patient who did not receive the Drinker respirator therapy was not very ill. He was frightened by the machine and fought it. There was only a partial involvement of the muscles of respiration and a unilateral pharyngeal paralysis. This patient recovered.

The pathological lung changes in patients who have expired in the respirator have always been of great interest to those taking care of poliomyelitis patients. The following changes are commonly reported in the literature:[9] (1) rupture of the alveoli due to emphysema; (2) pneumothorax; (3) pulmonary atelectasis; (4) pulmonary congestion; (5) pneumonia.

Most of these conditions can be eliminated in the spinal respiratory cases if the machine is used properly and the patients are systematically turned and carefully nursed. However, those with the more severe bulbar involvement die and at necropsy reveal changes such as were found in our group, namely: pulmonary edema, bloody fluid in the pleural cavities, subpleural petechial hemorrhages, and hemorrhagic bronchopneumonia.

The experience gained from our small series of cases has confirmed the observations of other writers who state that those unfamiliar with respiratory failure in poliomyelitis are apt to overestimate the value of the respirator in this disease because they fail to differentiate the types of respiratory failure. The spinal respiratory case has a very good chance of being saved by the machine. On the other hand, in the bulbar type with glosso-pharyngeal and vagus involvement the respirator often offers little help. Tables III and IV emphasize still more the importance of the foregoing statements.

TABLE IV. SUMMARY OF TREATMENT OF POLIOMYELITIS CASES WITH RESPIRATORY DIFFICULTY

University and Minneapolis General Hospitals, July 1, 1937–Dec. 31, 1937

Type of Case	Treatment	Total Cases	Recovered	Died
Spinal respiratory with paralysis of intercostal muscles and diaphragm	A	8	8	0
	B	0	0	0
Bulbar with pharyngeal and/or laryngeal paralysis	A	2	0	2
	B	13	12	1
Bulbo-spinal with paralysis of respiratory muscles and pharyngeal paralysis	A	5	0	5
	B	1	1	0

A—Cases treated with the Drinker respirator.
B—Cases in which the Drinker respirator was not used at any time.

Summary and Conclusions

Recent small epidemics of acute anterior poliomyelitis in Minnesota appear to reveal an increasing incidence of cases with respiratory difficulty. The respirator devised by Drinker and McKhann has been found to be of great help to the patients with difficult breathing. However, all patients are not benefited to the same extent.

Proper selection of the patients is essential. This is especially important in epidemic periods when the number of Drinker respirators available is less than the demand. Our experience with a small number of cases has shown the respirator to be of unquestionable value in the treatment of those patients with high spinal involvement. The machine gives them the rest so essential for recovery.

The Drinker respirator is of little or no value in the care of patients whose respiratory difficulty is bulbar in origin. The machine may overpower the choking and cough reflexes and in some cases even cause forcible aspiration through the larynx of secretions from the throat.

The bulbo-spinal cases may respond to the Drinker respirator provided the pharyngeal paralysis is unilateral. If the paralysis is bilateral and the patient cannot swallow, the machine is ineffective.

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SYPHILIS IN THE TRANSIENT*

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DURING the past few months you have noticed in the press or have heard over the radio considerable discussion about syphilis and gonorrhea. A campaign has been launched for the dissemination of knowledge in regard to syphilis, similar to the educational program regarding tuberculosis which has been actively carried on for many years. There are many reasons why the time seems appropriate for the carrying out of such a program, and I shall discuss this shortly. It is worthy of comment, however, that in the years gone by the editors of newspapers believed that their readers were not ready for such a campaign and that they could not use the words "syphilis" and "gonorrhea" in the public press. This attitude has gradually changed, and newspapers and magazines now contain information in regard to the venereal diseases. Surgeon General Parran of the United States Public Health Service precipitated the idea of such a campaign and has enthusiastically established it as a nation-wide enterprise. That the people of this country are in the mood to receive information about venereal diseases is evidenced by the fact that the momentum of the campaign has carried it from the hands of the Surgeon General to become a problem managed by the individual states.

There are numerous reasons why syphilis should be discussed in public rather than behind closed doors, as has been the case heretofore. We now have data based on extensive research which permits us to say without reservation that early

*From the Department of Dermatology and Syphilology, The Mayo Clinic, Rochester, Minnesota. Read at the Midwest Conference on Transiency and Settlement Laws, Saint Paul, Minnesota, March, 1937.

syphilis is definitely curable. I should like to emphasize the fact that cure applies to early syphilis. When we speak of late syphilis, that is, when the disease has passed the early stage, we are compelled to speak of an arrest of the disease rather than cure. By "arrest" we mean checking the progress of the disease in the heart or other internal organs. We know, now, that we can cure 85 per cent of the patients who come to us with a fresh infection and follow out the course of treatment which is recommended. It is by proper treatment of early syphilis that the late complications of the disease can be avoided.

Another reason why we are ready for a program of publicity is the success of such undertakings in Europe. The campaign in Denmark has been highly successful. There were less than 200 new cases of syphilis last year in Denmark, and I believe that in about thirty of these cases the patients were sailors who apparently had acquired the disease in some foreign country. In England, likewise, the campaign has been effective and has materially reduced the number of new cases of syphilis in that country. This decrease in the number of new infections is due not only to the fact that the treatments are furnished free but rather to the more important stipulation that the patient *must* remain under treatment until released by his physician.

A significant point for consideration is that in most cases of syphilis and gonorrhea the disease is acquired between the ages of seventeen and thirty-two years. Youths at this age are not impressed with the seriousness of the disease and, in most instances, are unable financially to arrange for proper treatment; this fact is, no doubt, a not infrequent cause of transiency. These boys are afraid of transmitting the disease to some of the relatives and because of their embarrassment and shame start out "on the road," spreading the disease as they go. Not only is it necessary that treatment be made available to these patients, but it must be made compulsory so that they will be forced to remain under treatment until they are no longer infectious, or able to transmit the disease.

Syphilis is infectious only during the early phase of the disease and is seldom infectious after the fourth year of its existence, regardless of whether the patient has received treatment or not. In other words, if an individual has had syphilis for more than five years and has been treated, there is practically no danger of his spreading the disease. In a man who has acquired syphilis and has had some treatment and has married five years after he acquired the disease, the likelihood is that his wife will not be infected and accordingly his children will not have the disease. However, if he marries within the first five years after he acquired the disease, the chances are that he will infect his wife, who in turn will infect the children, since the disease is transmitted to the children by the mother. This limitation of the infectiousness of the disease makes it an ideal disease for a public health program. We can absolutely eliminate the problem of infectiousness in cases of early syphilis by the adequate treatment of the patient. Treatment for two weeks will so heal the acute lesions that the infected individuals cannot spread the disease even though they will not be cured during this short time. We cannot do this with tuberculosis or other diseases of an infectious nature. Syphilis, therefore, lends itself to a universal health program because it is an ideal disease for the application of successful public health measures.

When I was first asked to discuss my impressions of the medical needs of transients, I was rather chagrined because I had had no contacts with transients. I did, however, have access to some reports on the incidence of venereal diseases among transients. It is unfortunate that no definite or concrete program was established in regard to venereal diseases early in the plans for the care of transients. The statistics which I was able to gather were from scattered sources and their interpretation was influenced by the enthusiasm of the medical officers who happened to be in control of the various areas. The United States Public Health Service at their Venereal Disease Clinic at Little Rock, Arkansas, took care of 1,125 patients with syphilis in 1934. Eight hundred forty-nine of these were on relief and 276 were self-supporting. Of these patients, 65 per cent were between the ages of sixteen and thirty years; 24 per cent had acute syphilis, 63 per cent had latent syphilis, and 13 per cent had late syphilis. The 63 per cent who had latent or dormant syphilis may or may not have been in need of treatment. They were not, however, in an infectious state and hence were not a menace to the community. They may have been in need of care and perhaps room and board, but

they were not of particular danger to anyone. Thirteen per cent had late syphilis, that is, syphilis of the heart or liver or nervous system. These patients were in need of treatment, perhaps in need of hospital care, to keep them from becoming state or county charges.

I have emphasized this particular group of figures because they represent incidence of damage from syphilis as it affects the populace as a whole. We know that less than a third of patients who have syphilis are going to have serious trouble from the disease, while two-thirds of those infected will have little if any trouble from the infection.

In Baltimore, there were 55,000 transients, of whom slightly less than 10 per cent had syphilis. Among the colored males, however, the incidence of syphilis was 23 per cent.

The Emergency Relief Bureau of New York City examined 40,000 men, of whom 3,100, or 8 per cent, had syphilis.

The report from Minnesota was obtained from two sources: Camp Independence and the Medical Health Center in Minneapolis. Camp Independence, during the three years of its existence, reported to the State Board of Health, 1,023 patients who had venereal diseases—702 had syphilis and 321 had gonorrhea. Of the 702 syphilitics, 176 had acute syphilis and hence were in danger of transmitting the disease. I have not been able to obtain statistics on the incidence of gonorrhea that are worthy of discussion. At the Minneapolis center, a group of 2,783 men received special examinations for evidence of syphilis. Of this number, 308 had syphilis and 107 had a history of syphilis; in other words, 11 per cent had syphilis. The reports on gonorrhea revealed fifty cases of acute gonorrhea and 700 men who gave a history of having had gonorrhea in the years gone by. The incidence of syphilis among colored men was 35 per cent.

Thus far I have been endeavoring to establish the fact that syphilis among transients is not a new problem. Syphilis among transients has always been a problem to those of us who have been interested in the disease. We will be able to do more, however, with more money available. I say this, having in mind the greatest appreciation for what has been done by the medical profession and charitable groups who for many years have given of their time, effort, and even material things for these patients, all of which has been contributed without thought or hope of compensation.

From these reports we find that the incidence of syphilis among transients is only slightly higher than is the estimated incidence among the populace as a whole. The United States Public Health Service estimated that 10 per cent of adults in this country have syphilis. The reports indicate that in the transient groups in the large cities, in the South, and in the Northwest, the incidence of syphilis varies from 10 to 21 per cent among the white population and up to 37 per cent among Negroes. The incidence of gonorrhea could not be estimated.

Under the provisions of the Social Security Act, the United States Public Health Service has available money which may be used for a venereal disease campaign. The law specifies that this money be allotted to the various states according to population, special health problems, and financial needs, at the request of the state health officials supported by the governing state authorities. The tentative plan of the campaign against venereal disease, as outlined by Surgeon General Parran, has as its general theme the elimination of syphilis and gonorrhea by making proper treatment available to all who need it. Some of the features of this campaign are worthy of emphasis.

For example, we have no accurate statistics as to the incidence of syphilis, so one of the first features of the program will be to require that all patients who have syphilitic disease be reported to the state boards of health. The reporting of these patients is a requisite in many states at the present time. A second feature is that treatment for syphilis is to be available, free of charge, to anyone who seeks it. In some states the Health Department will furnish to physicians the necessary remedies, such as salvarsan, bismuth, and mercury, on request. The number of venereal disease clinics is sufficient to give adequate care to patients in the majority of the urban centers, but in the rural districts such provisions are lacking and it will be necessary to create some method whereby the patients in these communities will be treated and the doctor who cares for them will be paid for his efforts. A fourth important point in the venereal disease campaign will be the organization of a group of field workers by the state health departments. It will be the duty of these workers to trace all

contacts, to arrange for the care of pregnant women who have syphilis and for the care of children who have congenital syphilis, and to locate and return for treatment patients who have had acute syphilis but who have discontinued treatment. When a diagnosis of gonorrhea or syphilis is made by a physician and treatment instituted, his problem does not end there but must include an effort to examine the individuals exposed to this patient. The success of this campaign will, in a great measure, depend not only on the efficiency of this "contact corps" but also on the creation of laws that will require these patients to remain under treatment until they are no longer a menace to their neighbors.

A fifth important item is provision of hospital beds for these patients. Patients who have acute syphilis should be isolated until they have been rendered noninfectious. The patient who has syphilis of the heart or liver, or late syphilis of the nervous system, may also be in need of a hospital bed, and just as much so as any other patient in the hospital.

I wish to emphasize also the effort to standardize the state laboratories where the diagnostic tests for venereal diseases are carried out. This is a technical detail which is already being worked out.

Finally, we must disseminate by the press, in pamphlet and in magazine, and by radio, knowledge regarding these diseases. A program for the postgraduate training of physicians in this field, under the combined direction of the state medical society, the public health service, and the American Medical Association, is also essential to the success of this plan.

What, then, are the medical needs of the transient who has a venereal disease? May I call attention again to the fact that venereal disease among transients is not a new problem to those of us who have been concerned with venereal diseases for these many years. True, we may have a different name for these patients, but the problem is no more acute. The evidence clearly indicates that the incidence of venereal disease among the unemployed is lower than it is among a low wage earning group. In the State of Minnesota, the patient who has venereal disease and is in need of treatment has not been neglected if he has sought aid. True, if neglect was evident, it occurred particularly in the rural areas, or if it occurred in the cities it resulted from the refusal of the patient to continue the treatment recommended by the clinics. I do not believe that anyone in the State of Minnesota who was in need of treatment for syphilis and applied for it was ever refused such treatment. In other words, I am endeavoring to establish the fact that there is no need for undue excitement or extreme measures to combat a problem that has been with us for a long time and that has in this state been rather well taken care of by the medical profession and the health department, in accordance with the funds granted them and abetted by the generosity of those who have given of their time and efforts gratuitously. The granting of additional funds to the State Health Department, which has been in direct contact with the problem, and has handled it very satisfactorily for the last twenty years, will permit of the early and rapid establishment of additional personnel trained in the epidemiology and the treatment of these venereal diseases.

I have made mention of the fact that this is not a problem involving only the transient; it involves indirectly or otherwise the entire populace of this commonwealth. The evidence indicates that the incidence of venereal diseases among transients in the Northwest is lower than it is in the South, due no doubt to the fact that venereal diseases are more common among the colored race. It is shown from the statistics gathered in this state that about 10 per cent of the transients will have syphilis, and that of this number a third will be in need of active treatment and two-thirds will have the disease in mild form. This last group is not infectious and consequently not a menace to the community. Treatment is given in the early cases with the idea of cure and of preventing the patient from spreading the disease. In the late cases it is given for the purpose of arresting the disease, extending life expectancy, and maintaining the individual's capacity as a wage earner. I have been informed that there will be some 6,000 or 7,000 unemployed men in this state during the coming summer months, of whom approximately 700 will have syphilis. Of this number approximately 250 will be in dire need of treatment. If this treatment is to be effective and if the patient is to derive benefit of a worth-while nature, regulations must be enacted to compel him to remain under treatment until dismissed or at least released as noninfectious. The experience of the

United States Public Health Service has shown that patients who have early syphilis have continued treatment on an average of six weeks, whereas cure in early syphilis is not possible before six months of continuous treatment, as an absolute minimum, has been given. The entire campaign will fail if measures are not at hand to compel these patients to remain under treatment until they are cured. The transient who has venereal disease must be cared for either in a transient camp or in an institution already provided with equipment and personnel for the care of these diseases, or by the local physicians. The granting of additional funds to these already established agencies will readily provide for care of the transient in the urban areas. The occasional case encountered in the rural communities can best be treated by the local health officer or physicians.

The medical profession of this state has, through a duly appointed committee,* agreed to encourage and work wholeheartedly in the campaign against venereal diseases. The Minnesota State Board of Health has played an active part in combating the disease by the treatment of these patients. With additional financial help they could reëstablish and maintain treatment facilities adequate not only for the transient but for the people of the state who need such medical care. In closing, may I emphasize that the transient who has a venereal disease is, after all, as he has been for many years, the problem of the health department. He is merely one part of the entire venereal disease problem in the state, which, if it is to be cared for efficiently, should be handled by a unit of the health department which is organized, experienced, and ready to cope with the entire problem in this commonwealth.

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CHORIONEPITHELIOMA WITH REPORT OF A CASE*

Survey of Incidence in Saint Paul Hospitals

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CHORIONEPITHELIOMA is a rare malignant tumor arising from the chorionic epithelium of the fetal villi following hydatidiform mole, abortion, and term pregnancy. The tumor may develop before the products of gestation have been expelled and has been described as occurring in the tube and ovary. Sänger, in 1889, first recognized the disease and applied the term "deciduoma malignum." Marchand, in 1895, correctly identified the tumor cells as derivatives of both layers of the chorionic epithelium and his views gradually became accepted. In 1898, he proposed the name chorionepithelioma.

Individual reports differ as to the incidence of chorionepithelioma. In a period of eighteen months, seven cases were found in 2,700 autopsies in Vienna. According to Curtis,[3] Symmers at Bellevue found none in 12,000 autopsies. Curtis,[4] in a coördinated clinical experience with Watkins covering sixteen years, found none, although constantly on the lookout. As noted by Gough,[8] Kimbrough (1934) observed two in 8,375 confinements; Winter (1934), three in 8,000 confinements; St. Sommer (1934), one in ten years among 18,000 confinements; and Joravieff (1933), one in 26,000 confinements. Sherman[17] (1935) in a thirty-four year study of hydatidiform mole at the New York Lying-in Hospital found one in 182,119 obstetrical and 14,280 gynecological patients. Schumann[16] (1937), in an analysis of its frequency in Philadelphia over a five-year period covering 207,707 pregnancies, found fifteen, or one in 13,850 pregnancies. Cosgrove[2] (1938) reported three developing in 20,450 confinements at the Margaret Hague Maternity Hospital.

The true etiology of this disease is unknown. In all but rare exceptions pregnancy has preceded the growth. Teacher[18] reported a series in which the age incidence ran from seventeen to fifty-five years, averaging thirty-three. The majority occur in multipara. It is a disease of the child-bearing years and of fertile women, with

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the incidence paralleling the degree of fertility. In Teacher's[18] monograph he found 8 per cent with the first pregnancy, 15 per cent with the second, and 28 per cent with the second and the third. Lynch and Maxwell[11] report the statistics of Briquel as 21 per cent occurring in the second pregnancy, 20 per cent with the third, and 47 per cent with the fourth or more.

Antedating so many chorionepitheliomas, hydatidiform mole has been considered as a predisposing condition. From Polloson and Violet's series of 455 cases, as reported by Curtis,[3] 45 per cent were found occurring after hydatidiform mole; 30 per cent after abortion; 21 per cent after labor at term; and 2.5 per cent followed ectopic gestation. In a series of 500 cases of hydatidiform mole, Findley[7] stated 31.4 per cent were followed by chorionepithelioma. Bell[1] gives the statistics of Sunde as 44 per cent occurring after hydatidiform mole; 30 per cent after abortions; and 22 per cent after labor at term. In 1922, Novak[14] estimated one per cent of moles became malignant and Teacher[18] believes five per cent too high. Polak[15] (1931), in discussing a paper on choriomas by Schmitz, stated that in ten years he had seen forty or fifty cases of moles and none became malignant and in the same interval ten chorionepitheliomas were not preceded by moles. In Schumann's[16] report, eight of the fifteen chorionepitheliomas were preceded by moles, while in Cosgrove's[2] analysis all three cases developed after molar pregnancies. With the incidence of malignancy following mole as 5 per cent or more, this in itself must be considered a grave condition.

The location of the tumor is most commonly in the area of the placental site. Grossly it invades the uterus as a soft, vascular mass, usually differentiated from the mucosal layer. It may be located on the endometrium, in the myometrium, or may grow through the wall and protrude into the peritoneal cavity or broad ligaments. The tumor, microscopically, is composed of masses of syncytium and nests of Langhans cells independent of villi with penetration into the wall of the uterus. There is invasion of blood vessels, mitosis of Langhans cells, very little stroma and intravillus vessels or lacking entirely. Hemorrhage and necrosis is common. The two types of cells vary in proportion in different tumors. Attempts have been made to classify these tumors. Mathieu and Palmer[12] and Gough[8] prefer Ewing's classification into: chorioadenoma, choriocarcinoma and syncytial endometritis. It is considered one of the most malignant and destructive neoplasms known. The reported mortality rate varies from 10 to 60 and 70 per cent. Findley[7] states there is a higher recovery rate from chorionepithelioma following mole than from that following abortions and full term pregnancies.

The period of latency varies from a coexistent appearance with the pregnancy to months and years. Usually it follows in weeks or months but may occur any time thereafter during the life of the patient. Metastases occur by way of the blood stream. Lesions in the lungs and vaginal walls are usually the first to appear.

Lutein cysts of the ovaries are regularly observed as in hydatidiform mole. They are usually bilateral and vary from small multiple cystomata to large tumors. These are probably due to excessive hormonal influence with stimulation of follicle growth and luteinization. Several investigators have shown that they are the result of excessive pituitary stimulation.

The usual clinical symptom is persistent bleeding following abortion, molar pregnancy or full-term pregnancy. The greatest difficulties are encountered in early abortions, where the bleeding may be thought to be due to placental remnants, and after labor wherein the physiologic bleeding may obscure the development of the growth.

The clinical value of the Aschheim-Zondek (1928) reaction for the study of normal and abnormal pregnancies has been well demonstrated. This biological test for anterior pituitary-like hormone is now an established aid in the diagnosis of chorionepithelioma. A number of authorities have shown that in hydatidiform mole and chorionepithelioma the amount of gonadotropic substance is much greater than that excreted during normal pregnancy. Due to the abnormal activity of the chorionic elements in these conditions the hormone secretion is increased to such an extent that even high dilutions result in positive reactions. On the basis of a quantitative reaction both the Aschheim-Zondek test or the Friedman modification may be used as a diagnostic aid. Ehrhardt[6] obtained a positive reaction in a case with 1/520 c.c. of urine and in another with 1/260 c.c. Levanthal and Saphir[10] reported 330,000 mouse units per liter of urine

in an early case and Kurzrok[9] observed a reaction as high as one million mouse units.

As recorded by Mazer and Edeiken,[13] in 1929 Fels and Roessler made several observations, namely: that with hydatidiform mole and chorionepithelioma much higher quantities of prolan are secreted than in normal pregnancy; the quantitative estimation of the hormone is an accurate guide in differentiating both of these conditions from uterine bleeding in the course of pregnancy due to other causes; presence of increased hormone for longer than two weeks after normal pregnancy or more than eight weeks after a mole pregnancy is pathognomonic of chorionepithelioma; continued presence of the hormone after the complete removal of the growth is indicative of metastasis.

Only living chorionic tissue causes positive reaction and the reaction becomes positive before clinical diagnosis of metastasis can be made. Mathieu and Palmer[12] diagnosed two early cases with the history of a mole and the persistence of anterior pituitary-like hormone in the urine. If a positive reaction should recur after having been reported negative following the expulsion of a mole, care should be exercised in differentiating between a new pregnancy and chorionepithelioma. With the advance of pregnancy, prolan concentration increases and twin pregnancies show a higher titer than single. Herein the quantitative biologic test lends a most definite aid, but should not be the determining factor to the exclusion of the history and clinical findings.

In the opinion of a number of investigators the biological test is more dependable than the findings from examination of uterine scrapings. Histological diagnosis from curettings is not always easy and may be inconclusive or even misleading. To establish the diagnosis by this method the operator must obtain a portion of the growth invading the wall of the uterus. Mistaken diagnoses have resulted in delay of proper treatment or the unnecessary removal of pelvic organs. Curettage is unreliable in that the growth may be located away from the uterine cavity in the myometrium. Levanthal and Saphir[10] (1934) and Cosgrove[2] (1938) each reported a case in which curettage would have been of no value. Moreover curettage carries the danger of perforation, infection, and possible dissemination.

Two reports have been made on a negative Aschheim-Zondek in the presence of chorionepithelioma: one by Fahlbusch in 1930, related by Mathieu and Palmer,[12] and another in 1937 by Schumann.[16] Schumann[16] believes radical operation is indicated whenever persistent fetal elements are found, especially if bleeding occurs, in spite of negative Aschheim-Zondek reaction.

The degree of malignancy cannot be predicted, and the prognosis depends on early recognition. The hope of curing any malignant disease depends on recognition in its early stage of proliferation. It is therefore advisable to have repeated Aschheim-Zondek or Friedman tests made in all women who continue to bleed after the expulsion of a mole, or in those with abnormal bleeding of an undetermined cause following pregnancy. Titus[19] suggests performing the test every two weeks until negative, and thereafter every four weeks for six months. Mathieu and Palmer[12] recommend monthly tests for one year.

Upon the diagnosis of this disease the early removal of all tissue containing malignant elements would be the ideal treatment. The clinician is then confronted with the question as to which type of operation offers the best hope of cure. The usual procedure has been to perform an abdominal panhysterectomy with removal of both tubes and ovaries. The abdominal operation permits better exposure and there is less trauma to the tissues. Lynch and Maxwell[11] advised the most extensive removal that the individual case will stand. Mathieu and Palmer[12] believe that in chorionepithelioma diagnosed early and the growth confined to the uterus it is unnecessary to remove the ovaries if they appear normal. They report an early case with only supravaginal hysterectomy and no recurrence after two years. Gough[8] maintains the lutein cysts are due to the disease and of no causative significance, and that the removal or conservation of the ovaries is optimal. He recommends the removal of the cervix. Total hysterectomy was the method of choice in Cosgrove's[2] three cases and no recurrences had appeared after 26, 23 and 11 months.

Few reports have been made of treatment by irradiation, its use having been limited chiefly to inoperable cases and metastatic growths. Davis[5] (1936) reports its successful use following supravaginal hysterectomy in a case complicating a placenta previa. In his opinion operable cases should be preceded or followed by thorough radiation. Titus[19] recommends preliminary radia-

tion with radium, followed by hysterectomy and a subsequent course of deep x-ray therapy. Irradiation should be effective as the growth is of fetal origin and such tumors are particularly susceptible to this kind of therapy.

The records of six representative hospitals in the city of Saint Paul were reviewed to determine the local incidence of chorionepithelioma.

Hospital A, 1928 to 1937 inclusive, reported 430 abortions, 6,310 confinements, one mole—no chorionepithelioma.

Hospital B, 1927 to 1937 inclusive, reported 589 abortions, 4,941 confinements, one mole—no chorionepithelioma.

Hospital C, 1928 to 1937 inclusive, reported 270 abortions, 2,995 confinements, one mole—no chorionepithelioma.

Hospital D, 1932 to April, 1938, inclusive, reported 1,279 abortions, 8,150 confinements, seven moles—no chorionepithelioma.

Hospital E, 1932 to April, 1938, inclusive, reported 365 abortions, 3,705 confinements, three moles—no chorionepithelioma.

Hospital F, December, 1920, to April 7, 1938, inclusive, reported 330 abortions, 7,282 confinements, four moles—no chorionepithelioma.

In a total of 3,263 abortions, 33,383 confinements and 17 moles (36,646 pregnancies in all), there was no record of a chorionepithelioma.

It should be stated that one of the hospitals reported one mole and four chorionepitheliomas. However, upon investigating two of these charts and the pathological reports, they were omitted as entirely inconclusive. Of the remaining two, prepared sections of the surgical specimens were submitted to Dr. E. T. Bell for examination. Dr. Bell reported no chorionepithelioma present.

Report of Case

The following constitutes the report of a chorionepithelioma of the uterus antedated by the expulsion of an hydatidiform mole.

Mrs. J. S., thirty-four years of age, married at twenty-two, had five children living and well. There was a history of a spontaneous miscarriage at four or five months in March, 1934, unattended by a physician. Menses were regular until mid-summer, the last menstrual date not being definitely known. She first consulted her physician August 16, 1934, complaining of a tired, worn-out feeling and nervousness. His examination showed her weight to be 142 pounds, blood pressure 128/75, and hemoglobin 70 per cent. She was again seen August 30, 1934, and under medication her hemoglobin had risen to 80 per cent and she felt some better. On September 24 she reported again with the complaint of slight uterine bleeding, but no associated cramps. Examination revealed a uterus about two and a half months pregnant. The patient was put to bed for one month, including one week of hospitalization. The bleeding recurred upon being upon her feet. On November 7 abortion was induced by the bougie method after consultation with another physician. The induction was successful and a mass the size of a fist was expelled, described as meaty in appearance. The specimen was examined by Dr. E. T. Bell and reported as hydatidiform mole. Moderate bleeding continued for three weeks and several particles of tissue were passed. On November 30, 1934, the physician performed a dilatation and curettement with the removal of particles of vesicular tissue. This tissue was not examined. December 19, 1934, the uterus was of normal size and no bleeding had occurred. January 2, 1935, the patient reported to her physician that bleeding had recurred that day.

The patient was first seen by me in consultation January 4, 1935, with the complaint of uterine bleeding of two days' duration, and slight lower abdominal cramps. The amount was described as slightly in excess of the usual menstrual flow. There had been no evidence of bleeding for thirty-three days following the dilatation and curettement.

The family history was essentially negative.

Examination showed a well developed woman of 138 pounds weight, temperature 97.2, pulse 80, blood pressure 118/72, chest and abdomen normal. Pelvic examination: vulva, urethra, and glands negative; perineum somewhat relaxed; vaginal walls normal in appearance; old healed lacerations of cervix with chronic cervicitis present; fundus retrodisplaced, uniform in outline with the suggestion of being slightly enlarged; nothing palpable in either adnexal region. Laboratory findings: urine normal; hemoglobin 75 per cent; red blood count 4,000,000; white blood count 10,500.

Bearing in mind the history of the mole, it was, nevertheless, assumed that this was her first menstrual period following the dilatation and curettage, and retrodisplacement and slight subinvolution of the uterus was determined. It was suggested to the patient's physician that hormone tests for the presence of persistent hydatid reaction be made and that she be observed for the possible development of malignancy. On January 26, 1935, a Friedman test was positive. This was an undiluted specimen. The physician reported that she was continuing to have a slight bloody discharge off and on. February 27, 1935, a Friedman test was again positive. Through a misunderstanding the request to the laboratory for dilution of the urine was omitted. Her physician reported that irregular bleeding continued and the uterus was enlarged.

The patient was again seen by me March 12, 1935. Bloody vaginal discharge had continued at intervals. General examination was much the same as on previous visit. Her weight remained the same and the temperature was normal. Pelvic examination: inspection of vulva and vaginal walls showed no implants; thin bloody discharge from cervix; uterus definitely enlarged to the size of a 3 to 3½ months pregnancy, quite uniform in outline and slightly tender; adnexæ

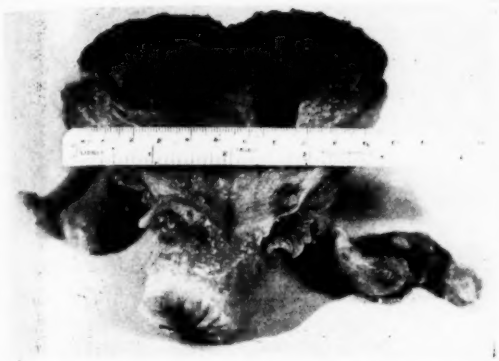

Figure 1. Photograph of gross specimen showing the entire uterus and its appendages with the anterior surface incised to expose the tumor in the fundus.

not palpable. Laboratory findings: hemoglobin 72; red blood count, 3,870,000; white blood count, 11,200; differential—lymphocytes, 33, large mononuclear 1, neutrophiles 65, eosinophils 1 per cent. Sedimentation rate moderately rapid, Wassermann negative, blood grouping 11. X-ray of chest negative for metastases. A clinical diagnosis of chorionepithelioma was made and operation advised.

On March 15, under spinal anesthesia, the abdomen was opened. The uterus was enlarged to the size of a three and one-half months' pregnancy. The growth, confined to the fundus and slightly nodular on the serous surface, presented a striking multicolored appearance. The ovaries were only slightly enlarged and cystic. A panhysterectomy was carried out, with the removel of both tubes and ovaries.

The uterus and its appendages weighed 320 grams. The inside diameters of the tumor measured roughly 5 by 7 cm. The pathologic report made by Dr. E. T. Bell of Minneapolis was chorionepithelioma of the uterus; numerous follicular and luteal cysts of the ovaries.

The patient made a good recovery and was discharged from the hospital March 29, 1935, on her fourteenth postoperative day. Deep x-ray therapy consisted of a total of 143 per cent skin erythema dose to the pelvis in ten treatments over a period of eighteen days, through four portals.

No immediate biological tests were made on this patient following surgery and radiation. The follow-up of this patient has been restricted to personal communications. The first one of these was received in July, 1936, in which the patient stated she had gained 17 pounds in weight, and, except for being a little nervous, felt well and worked hard every day. Recent communications in the last few weeks from both the patient and her physician were to the effect that she remains in good health and there is no evidence of recurrence. I believe that she may be considered cured.

Subsequently, on May 12, 1938, a Friedman test was negative.

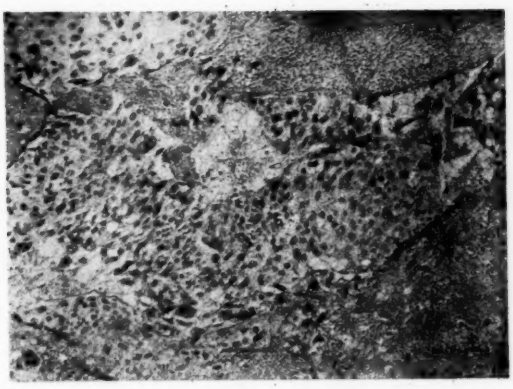

Fig. 2. Photomicrograph showing masses of chorionic epithelium in the uterine muscle independent of villi. The tumor cells are largely the Langhans type, but there are a few syncytial cells at the periphery of the section.

Summary

1. The subject of chorionepithelioma has been reviewed.

2. The incidence of chorionepithelioma among 36,646 pregnancies and abortions in six Saint Paul hospitals has been given, substantiating the reported rarity of the condition.

3. A case report has been presented.

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SPINA BIFIDA CYSTICA OF THE PELVIS: DIAGNOSIS AND SURGICAL TREATMENT*

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A PATIENT who recently came to The Mayo Clinic because of spina bifida cystica of the pelvis presented an unusual problem for diagnosis because of the location and rarity of the lesion and also because a new operative procedure was required. This experience I believe justifies a report of the findings and the surgical technic employed. Since there still remains some confusion about the types of operations to employ for spina bifida, and some uncertainty about the proper time to operate, I shall review our experiences in the general discussion of the subject.

Spina bifida is one of the common deformities of the newborn; it occurs once in 1,000 to 2,000 births. There are two varieties. One is known as spina bifida cystica and the other as spina bifida occulta. The spina bifida cystica has three subdivisions. The first is simple meningocele, which represents a herniation of the meninges through a defect in the spinal canal. The cystic wall is composed of arachnoid and dura, which are covered with thinned fascia and skin. The meningocele contains no nerve elements. The second variety is known as myelomeningocele. This is similar to a meningocele except that nerve roots enter the wall of the cystic mass. The third variety is known as a syringomyelocele. It resembles the first variety in that it is composed of a sac but differs from the first and second varieties in that it contains a dilated portion of the conus medullaris, and nerve fibers end blindly in the wall of the sac. The first variety is rarely associated with any form of paralysis; the second variety is associated with a varying degree of sensory or motor disturbance; the third variety usually is associated with very marked paralysis. At this point, I should like to emphasize that a plastic repair on the cystic mass rarely improves the paralysis. Therefore, the parents of the patient should be informed of this fact before operation is performed.

An associated hydrocephalus of the communicative type may accompany any one of the three varieties. However, it is most frequently associated with the third variety. If the two coexist, each requires separate surgical consideration. It has been observed that a repair of a large cystic spina bifida in cases in which there is no obvious evidence of hydrocephalus may result in a hydrocephalus as the spina bifida has served as a reservoir during the early weeks of life. Spina bifida may occur in any portion of the spinal canal. It occurs more frequently in the lumbosacral region than in other parts of the spinal column. However, it does occur in the craniocervical and in the cervical region, but it is rarely seen in the upper thoracic region. The spinal defect may include from one to ten vertebræ.

Spina bifida occulta is characterized roentgenographically by a bifid spinal column and by the appearance of a small dimple covered by a tuft of hair. It rarely produces clinical symptoms in early life, but the progressive myelodysplasia may cause symptoms later in life. This manifests itself by increasing sensory and motor disturbances and impairment of the rectal and vesical sphincters.

Analogous to the developmental defects of the spinal cord observed in the later stages of spina bifida occulta is a condition that is known as myelodysplasia, except that it occurs without a demonstrable bony defect.

Etiology

One of the oldest theories, strongly championed by Morgagni in 1779, rejected by most writers since von Recklinghausen's[12] comprehensive dissertation on spina bifida, in 1886, and recently rediscovered and espoused by numerous writers, is the theory of hydromyelia. According to this theory the choroid plexus, activated perhaps by a hormone, secretes such a large quantity of spinal fluid that it either prevents union of the medullary folds or ruptures them after union has taken place. Interference with the absorption of spinal fluid is advanced as the alternative mechanism. The rapid accumulation of fluid then is given as the primary cause; the resulting cyst is

*From the Section on Neurologic Surgery, The Mayo Clinic, Rochester, Minnesota. Thesis presented before the Minnesota Academy of Medicine, April 13, 1938.

interposed as a bulging mass between the lateral mesodermal structures, preventing the approximation and fusion of the lips of the medullary groove. One of the main supports of this theory lies in the fact that spina bifida is often associated with hydrocephalus, which is rapidly made worse or, if not already present, may rapidly be produced by operative closure of the spinal defect. The assumption of an excessive amount of spinal fluid obviously depends on the further assumption that the choroid plexus is secreting fluid at this early embryonic period. This hypothesis seems inadequate when the whole problem is considered; one of its most serious objections is the fact that the choroid plexus does not begin secreting fluid until about the tenth week, 2.4 mm. embryo (von Monakow),[9] whereas all writers agree that the spina bifidous deformity is produced no later than the third week. Further, the cases in which there is a failure of the entire neural canal to close certainly represent a more serious type of the same fundamental defect and must accordingly be explained on the same basis; it seems highly improbable that the amount of spinal fluid secreted is so immense that the entire canal, from its cephalic to its caudal ends, would be torn open or union prevented throughout by the flow of the choroidal secretion. Complete absence of the cord, or amyelia, which accompanies many of these severe types (Schmaus and Sacki)[13] must also be explained by a more comprehensive theory. Mention of micromyelia, diastomyelia, and diplomyelia, which occur with spina bifida and without it, would still further embarrass the theory. It is also irreconcilably at variance with the observation that the portion of the spinal cord involved lacks all evidence of pressure myelitis, with its disintegrating nervous tissue; on the contrary, it is seen to have remained at a standstill in its embryonic development, with embryonic nerve cells and embryonic blood vessels, as the area medullovasculosa (von Recklinghausen).[12] Associated defects, such as harelip, cleft palate, and club feet, are considered by recondite inference to be the results of pressure on the nerve centers. While it must be admitted that the added area of absorption supplied by the cyst often prevents the occurrence of hydrocephalus, it would seem more reasonable to suppose that the faulty absorption of spinal fluid may also be based on some developmental defect. An associated cardiovascular defect has been suggested as a possibility.

von Recklinghausen[12] believed that spina bifida is primarily due to a failure of the mesodermal envelope of bone and dura to approximate; he looked on the ectodermal dysontogenesis as secondary. The fact that a myelodysplasia of this type may occur without defects in the bone controverts this theory; it suggests that the defect may be primary in the medullary plate. The types of spina bifida cystica and spina bifida occulta not accompanied by defects in the nervous system argue for the reverse. The necessity for making one primary and the other secondary is not apparent. Whatever the exact mechanism may be, it does not complete our search for the more fundamental process in which we are primarily interested.

There is much evidence against assuming that the basis is germinal, or developmental, and such cause has been generally discarded. I do not believe that this is altogether justified. Thus, the presence of developmental defects in one case, in which the closely related defect of enuresis and sacral dimple were transmitted to six persons on the male side, through three generations, is clearly an instance of heredity. Two other cases, in which the same defect appeared in siblings, do not furnish conclusive evidence, since maternal environmental factors might have been just as potent and quite as likely as developmental factors. On the whole, the hereditary element hangs on a very tenuous thread; it must be assumed for occasional cases, but these are not numerous enough to argue for its acceptance as the sole cause.

Experimental Production of Spina Bifida and Other Anomalies

Recent work[17] among biologists cannot be disregarded as some writers are disregarding it simply because it has been carried out on lower forms of life. The experimental production of spina bifida by modification of the environmental medium has taught us a great deal. A brief review of this evidence is profitable. Probably the most striking demonstration of the importance of environment in the production of spina bifida was that produced by Hertwig,[5] in 1896, who subjected the axolotl, a salamander, to different concentrations of sodium chlorid solution. He found that a 0.5 per cent solution had no

effect, a 0.6 per cent solution produced monsters in 50 per cent, while 0.7 per cent solution resulted in development of spina bifidous monsters in every case. Stockard, using *Fundulus heteroclitus*, the common minnow, produced spina bifida by using magnesium chlorid. Cyclops could be produced in at least 50 per cent of cases, which was somewhat more frequent than spina bifida. It was also demonstrated that alcohol, ether, and the alkaloids could be used with similar results.

In order to demonstrate the applicability to man of these factors of monster production, Werber used substances produced in the human metabolism, namely butyric acid and acetone. He exposed *Fundulus heteroclitus* in the two, four, eight, and sixteen cell stages, to the action of 1-12/1-14 gm. molecular solution of butyric acid in sea water, from fifteen to twenty hours; he produced a great variety of monstrosities, the extreme defect being the development of only an eye or an ear, the rest of the embryo failing to appear. Higher percentages of acetone killed the embryos, while lower concentrations resulted in the development of monsters. Werber[15,16] concluded that faulty maternal metabolism might well be the underlying cause of dysontogenesis. Disastrous maternal effects of diabetic patients are well known. Keibel and Mall[6] found that the chorion of nearly all monsters had been the seat of inflammatory processes which in time might well have interfered with normal metabolism. The frequent association of hydramnios makes this all the more probable.

Chemical methods are not the only ones by which monsters have been produced artificially. One of the other simplest devices is a modification of gravitational forces on frogs' eggs. Simply turning upside down (Conklin)[4] frogs' eggs in the two-cell stage may cause the development of double-headed or double-bodied monsters. A redistribution, by centrifugalization, of the heavier elements of the eggs constitutes another method. In the ascidian (sea squirt) eggs, in which different kinds of protoplasm give rise to different organs and tissues, this rearrangement may result in marked dislocation of organs. That the problem is complex is shown by the fact that some varieties develop normally in spite of this artificial rearrangement (Morgan).[10]

Lewis[8] adopted the remarkably simple method of destroying different portions of the eggs of *Fundulus heteroclitus* by operation-needling, and so forth, and demonstrated that the organs predetermined in the portion of the egg destroyed did not develop. He could thus produce a developmental defect in any portion of the body he desired. Kellicott found that subjecting the eggs of *Fundulus heteroclitus* to a temperature of the average household refrigerator, for a few hours or days, sufficed to produce every variety of defect.

That physicochemical action may produce developmental mental defects was demonstrated by Bardeen,[3] who exposed both male and female frogs to the x-ray for one hour, prior to fertilization. He learned that exposure to the x-ray of the ovum or the sperm sufficed to influence the subsequent development of the eggs in such a manner that marked abnormalities, including spina bifida, resulted. Baldwin[2] produced spina bifida in frogs by exposing a given part of the egg to the action of the ultra-violet ray.

The mechanism by which various factors influence the developing organism is also disputed. I can only refer to these briefly. Mall assumed that nutritional factors *in utero* resulting from diseased fetal members underlie monster production. Werber postulated the theory of blastolysis, according to which a part or wedge of the germ substance is destroyed, resulting in anomalous fusion or dispersion of the part split off. Stockard[14] believes that he has proved, by the use of magnesium with its well-known anesthetic or inhibitory action, that an inhibitory action is responsible. Kellicott,[7] espousing the hypothesis of disorganization, believes that this must take place before differentiation by gastrolysis, through interference with the organization of the fertilized ovum. The discovery of the deleterious action of the x-ray and radium on the sperm, prior to fertilization, added another complication, since it proved that an abnormal character of the gametes may in some instances suffice to produce the defect.

von Recklinghausen's demonstration that the area medullovasculosa contains elements retaining early embryonic characteristics is of fundamental importance and prohibits the acceptance of the current theories, in the strictest sense, as an explanation of the large group of cases not dependent on hereditary factors; or is it possible that lack of abnormality of function and metabolism, secondary to the apparent isolation of

these elements, in the area medullovasculosa suffices to explain their embryonic appearance.

In view of the foregoing clinical observations and the facts adduced through experimental methods, it seems that spina bifida cannot be explained on the basis of any single factor, but by one or more of the following causes: abnormal character of the gametes or mechanical, chemical or physico-chemical factors influencing the embryonic rudiments, either before or after differentiation; the mechanistic action of accumulated cerebrospinal fluid could act only as a secondary cause. A conception as broad as this seems to destroy all semblance of a theory; indeed, any precise formulation of a theory which does not take all these factors into account would be dogmatic, premature, and untenable at the present stage of knowledge.[17]

Symptoms

The physical deformity of a spina bifida cystica is obvious. The cystic masses vary in size from those which are similar to an English walnut to those which are larger than a grapefruit. The roentgenogram reveals the extent of the bony malformation. The neurologic examination reveals findings according to location and extent of the involvement of the nerves or spinal cord. It does not disclose any change in the presence of a meningocele, it shows but few nerves missing when a myelomeningocele is present and gives evidence of marked anomalies of the nerves and spinal cord if a syringomyelocele is present. Hydrocephalus[11] frequently accompanies the third variety but rarely is associated with the other two, and never is associated with a spina bifida occulta.

Surgical Considerations

Oftentimes surgeons are called upon to repair spina bifida cystica as soon as it is observed after birth. Occasionally, this can be done with success, but, judging from our own experience, it is evident that operations on babies carry a much higher mortality than do operations that are performed at a later date, let us say, at a period from six to nine months following birth. There are other advantages in waiting. First, an apparent hydrocephalus may not be evident at the time of birth but will be evident in six to nine months following birth. Second, neurologic findings are often difficult to demonstrate in a small child. Therefore, by waiting for six to nine months it becomes much easier to demonstrate neurologic findings. A third reason for delaying the operation is that the parents can observe for themselves whether or not an accompanying hydrocephalus exists and whether or not any paralysis is caused by the anomalous condition. They should be thoroughly informed of these findings and should also be instructed concerning the results of the operation. They should be told frankly that the operation consists of a plastic closure and that the operation will not alter the hydrocephalus nor will it restore any function to the paralyzed extremity. It is well to go into detail concerning the object of the operation. The parents should be told what to expect in the way of results. The two first varieties, namely, meningocele and myelomeningocele, lend themselves to surgical treatment, but the syringomyelocele rarely presents a true indication for surgical treatment. Occasionally, one finds it advantageous to repair the cystic mass, but little is accomplished by attempting to free the conus medullaris from the scar tissue. Occasionally, one is able to free nerve filaments in order that they may fall back into the lumbosacral canal. When this is possible there may be some subsequent improvement in that particular nerve.

If an accompanying hydrocephalus exists, it is usually of the communicative type. Occasionally, repeated spinal punctures and limitation of the fluid intake may control the situation. If the child is a particularly healthy one, the surgeon may be justified in performing a Tracy-Putnam operation. This operation includes two separate openings of the lateral ventricles, through which the choroid plexus is coagulated for the specific purpose of decreasing the amount of cerebrospinal fluid secreted. Unfortunately, neither procedure guarantees that the hydrocephalus will be controlled. Since it is impossible to guarantee a control of the hydrocephalus, there is danger that the plastic repair may not hold and that leakage of cerebrospinal fluid may follow; when this does occur, there is always the danger that meningitis may develop.

If the obstetrician, as well as the parents, have been convinced that the plastic repair of the spina bifida cystica should be deferred until the child is six to nine months of age, they both ask two questions: (1) "How shall the spina bifida be cared for?" (2) "In the event that it leaks, how shall the leakage be controlled?" In our

experience we have observed that the spina bifida can be protected nicely by the application of a doughtnut-like ring that is made of cotton and held in place with roller bandage. The ring can be fastened to the back with adhesive plaster. It serves as a wall about the cystic mass and thus prevents undue pressure on the mass if the patient is lifted or allowed to roll on his back. With this protection, the child can be handled just as any other child is handled during the early months of life. Frequently, abrasions or ulcerations are present on the thinned-out skin in the dorsal portion of the sac. When these occur, the macerated area can be treated with sterile petrolatum, or preferably sterile borated petrolatum. The crusts can be removed from time to time with pledgets of cotton soaked in liquid petrolatum which has also been sterilized. After the abrasion has healed, the cystic tumor should be washed daily with soap and water, and powdered with any good antiseptic powder. If a rupture has taken place, so that the cerebrospinal fluid leaks from the cystic mass, we find that a similar treatment may be employed, except that the child should be kept more or less in the prone position on a pillow, with the head slightly lower than the cystic mass, which prevents any hydrodynamic pressure on the sac itself. Occasionally, one is justified in aspirating the sac; this is done by sterilizing the skin at the base of the sac and introducing a needle through normal skin, directing the needle superficially into the midportion of the sac. Following aspiration of the sac, it is well to apply gentle pressure with a binder over a sterile cotton dressing. The cotton dressing never should be applied directly on a macerated area. It is much wiser to cover the macerated area with a strip of vaseline gauze before applying sterile cotton dressings.

The reason for waiting from six to nine months is that it allows one to evaluate the situation, to determine the degree of paralysis, if any exists, and to determine whether or not there is a coexisting hydrocephalus. It also allows the child to develop so that additional feedings of gruel and semisolid food can be given. The reason for not waiting longer than nine months is that the operation, if it is to be performed, should be performed before the child attempts to stand, for at this time he is very likely to fall onto the sac and thus press it unduly.

In selecting an operation, one should choose an operation that will permit a thorough exploration of the contents of the spina bifida before a plastic closure is made. It is unwise to employ the older technic, which consists of freeing the sac along its base, tying and transfixing it without an observation from within, as it may include a knuckle of roots or cord when in reality they have not become a part of the sac. There is no need to worry about a recurrence of the spina bifida cystica if there is no hydrocephalus. We have learned that it is unnecessary to employ any sort of bone graft, since a good plastic closure suffices to protect the nerves or cord in the spinal canal. This argument is substantiated by the fact that one never sees a spina bifida cystica result following a laminectomy for fracture of the spine or for the removal of an intraspinal tumor.

Spina Bifida Occulta

The symptoms that result from the associated myelodysplasia make their appearance in early adult life. They are characterized by deformity of the feet, disturbance in the reflexes, the appearance of sensory changes and the loss of control of the bladder and rectum. The symptoms increase as the age of the patient increases. Exploratory operation has been performed in a number of these cases. Some investigators have reported lipomatous masses which they believe have been responsible for the symptoms. Others have found hypertrophy of the ligamentum flavum, but unfortunately the surgical results have been rather disappointing, since the degeneration takes place in the conus medullaris whenever the lesion involves the lumbar or sacral region. Spina bifida occulta is not confined to the lumbosacral region and fortunately may exist without producing clinical symptoms.

Surgical Technic.—The patient is anesthetized while on his side.[1] Ether, administered by the drop method, is the anesthetic most commonly used. After the patient has been anesthetized, he is placed on his abdomen over a pillow with the head lower than the spina bifida cystica, in order that the cerebrospinal fluid will not drain from the ventricles when the sac has been opened. The skin is thoroughly cleansed with green soap, washed with ether or alcohol, and a solution of merthiolate is applied as an antiseptic. Following adjustment of the sterile linen, the sac is opened longitudinally in order to permit an internal exploration of the sac. If no nerve ele-

ments are present, a circular incision is made in the meninges peripheral to the defect in the spinal column, in such a manner as to permit an apposition of the meningeal flaps over the defect. Occasionally, it is advisable to reflect a flap of lumbar fascia across and suture to the opposite side in order to reinforce the closure of the meninges. After removing the redundant tissue the wound is then closed. It is preferable to use number 0 chromic catgut for the subdermal sutures. The skin is closed with interrupted silk sutures and the wound is covered with collodion dressings. Collodion dressings have the advantage of protecting the wound from contaminations of urine and feces. In the event that nerve elements have been found to enter the walls of the sac, it may be necessary to resect these along with the redundant tissue, but whenever it is possible to free nerve filaments or a portion of the cord and permit it to fall into the spinal canal, this should always be done. The freeing of adhesions oftentimes does permit a number of nerve elements and occasionally the cord to seek their former position, but even though this has been accomplished, too much credit should not be given to the operative procedure in the hope that additional recovery will take place in the paralysis. I have seen improvement in sphincteric control.

In the event that paralysis does exist as a result of the anomalous condition of the nerve and spinal cord, the relatives should be told that the situation might later be improved by orthopedic measures. In some instances, braces may be made. In other instances, fixation of flail joints may assist in the use of the extremity. The most serious sequela is the difficulty that is associated with incontinence of the bladder and rectum. The patient should be instructed to eat food that will regulate the bowels, and thus, by the aid of an enema, manage the daily evacuation. The patient should be instructed to develop automatic control of the bladder. Evacuation often can be initiated by a massage of the lower part of the abdomen. Urinals may need to be worn continuously. Catheterization should be avoided whenever possible. In a few instances, presacral neurectomy has proved of value in regulating evacuation of the bowels and bladder. In still fewer instances, lumbar sympathectomy has proved of value in relieving the vasomotor changes of the lower extremities, by improving the circulation and preventing ulceration.

Report of Case

A single woman, aged twenty-two years, whose father was a physician, first came to the clinic on September 4, 1935. Her chief complaint was pain that was situated in the region of the sacrum. The pain had first been noted when the patient was thirteen years of age. The appearance of the pain had been intermittent. At times it had been so severe that it had compelled her to go to bed for two or three days. She did not complain of a true sciatica, but said that the pain did extend to the left knee. The pain had no relation to the time of day. The patient had been told that she had some abnormality of the sacrum and that she had a tumor in the pelvis. She had not had any other symptoms and the previous history was not significant. Examination disclosed tenderness in the left lower quadrant of the abdomen and a cystic mass that was situated in the hollow of the sacrum. Orthopedic examination revealed a marked lumbar lordosis, in addition to some tenderness over the lumbosacral region and over both sacro-iliac joints. Pelvic examination disclosed no abnormality except for the palpable mass previously mentioned. Examination of the blood and urine did not disclose any abnormality. Flocculation tests were negative. The roentgenograms revealed an absence of the lower half of the sacrum (Fig. 1). This was explained as a probable congenital aplasia. A neurologic examination failed to reveal any abnormality. The cystic tumor was found on rectal examination. A tentative diagnosis of a dermoid was made, but inasmuch as the patient was not urged to undergo operation at her initial visit to the clinic, she returned home without any surgical treatment.

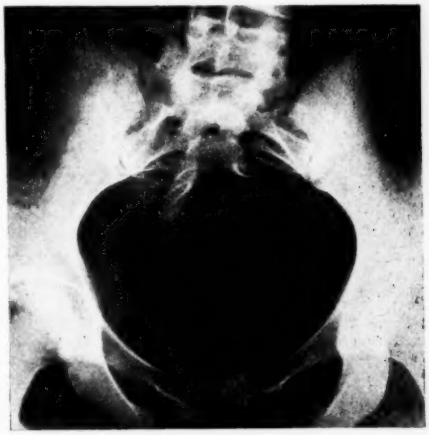

Fig. 1. Malformation of the sacrum caused by a spina bifida cystica that originated in the sacral region and extended into the pelvis.

The patient returned on July 21, 1936, with instructions to consult me in view of my interest in the treatment of sacral tumors. She then informed us that after

she had left the clinic in September, 1935, she had continued to have sacral pain. This had been so severe that an exploratory laparotomy had been performed elsewhere in October, 1935. A retroperitoneal cystic mass had been found in the hollow of the sacrum, posterior to the rectum. The mass had not been opened or removed. The abdominal wound had been closed, without any surgical intervention. In May, 1936, the patient had spent six weeks in bed because of a persistent fever. For a few months previous to her second admission to the clinic, she complained of persistent paresthesias which had involved the left foot and leg. At times, the pain had extended upward along her left side, but had not involved the face. She did not complain of any objective loss of sensation. Her menstrual history did not indicate any abnormality. She was free from all other symptoms except constipation, which had been troublesome at times. She had not noted any dysuria or urinary frequency. At times, she thought that her abdomen had swelled, at least it had appeared to be tender in the left lower quadrant. Urinalysis did not reveal any abnormality. Additional roentgenologic examination of the spinal column revealed the same finding that previously had been observed. The proctoscopic examination revealed nothing abnormal in the rectum or sigmoid colon. The proctologist observed a large, soft cystic mass which was situated posterior to the rectum. The results of neurologic examination again were negative. The ophthalmologic examination likewise did not reveal any abnormality. At this time, it became my good fortune to see the patient in consultation. I reviewed the history and the findings and proceeded to carry out a digital examination of the rectum. I could palpate a soft fluctuant mass, about the size of a kittenball, situated posterior to the rectum in the hollow of the sacrum. The mass was definitely cystic and was less firm than a dermoid or a chordoma. However, both of these tumors had to be considered in the differential diagnosis. I ventured the opinion that we were probably dealing with a spina bifida cystica which had resulted from an anomalous development of the sacrum, and I advised an exploration of the sacrum through a laminectomy performed over the third and fourth sacral vertebral segments.

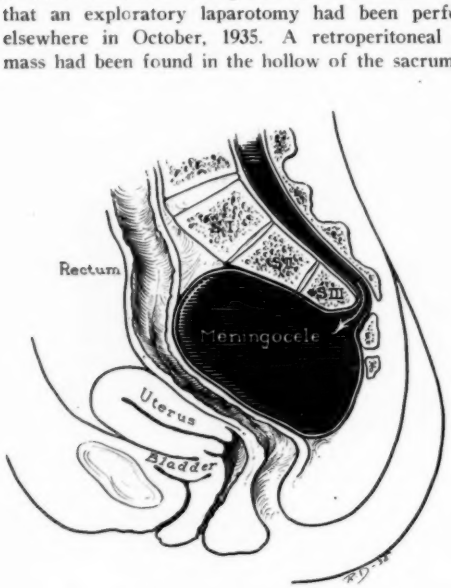

Fig. 2. Schematic illustration of a spina bifida cystica which was situated in the pelvis posterior to the rectum and in the retroperitoneal space.

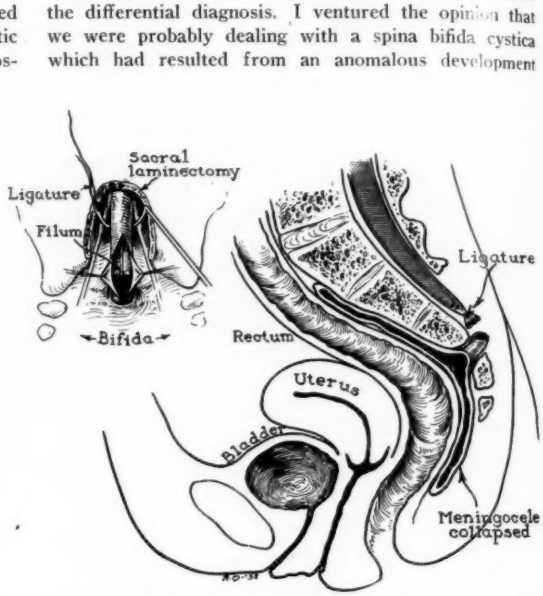

Fig. 3. Exposure of the caudal end of the sac through a small laminectomy wound, the resection and closure of the sac; this procedure permits the pelvic spina bifida cystica to drain into the soft tissues and collapse.

The laminectomy was performed opposite the third and fourth sacral segments of the vertebral column, through a dorsal midline incision. Upon exposing the dural sac, it was found to communicate with the pelvic mass. I opened this at the lower end of the incision and found that the cerebrospinal fluid in the subarachnoid space communicated with the fluid within the pelvic mass (Fig. 2). I then had to determine whether or not it contained nerve elements and found that the only structure that passed down through the dural sac into the pelvis was the filum terminale, which measured about 2 mm. in diameter. There were no nerve elements in the sac at this location. The nerve fibers had left the dural sac at a higher level.

The problem that confronted me was how to remove this sac, but it soon became apparent that it was impractical to attempt to remove the pelvic mass. Therefore, I decided to divide and ligate the dural tube, in the sacral canal, thus closing the caudad end (Fig. 3). This was accomplished by a purse-string suture and additional reinforcing ligatures. Upon exploring the pelvic mass I found that I could introduce a catheter for a distance of 5 inches. Subsequent exploration with the lighted retractor showed that the arachnoid lining of the dural sac was rather gray in appearance and free from any unusual masses. This convinced me that I was not dealing with a dermoid and that I was dealing with a spina bifida cystica which had extended an-

teriorly into the pelvis in the retroperitoneal space posterior to the rectum. After aspirating the cerebrospinal fluid in the pelvic portion of this sac, the walls were seen to collapse as a result of the increased intra-abdominal pressure. Thus, it occurred to me that if I left the distal portion of this sac open, it would automatically close itself and become adherent. Therefore, I proceeded to treat it in this manner. The wound was closed and the patient cared for very much as we care for other patients who have spina bifida. The patient remained under observation in the hospital for sixteen days and was dismissed from the clinic twenty-one days after the operation, which was on July 22, 1936. During her postoperative convalescence, it was necessary to catheterize her for the first five days. A postoperative neurologic examination did not furnish any additional information. The patient said that the pain was less severe than it had been but that she was still conscious of some paresthesia on the outer aspect of the left foot and around the saddle area of her buttock. A rectal examination on the day of her dismissal did not reveal any pelvic mass. It was impossible to palpate any sort of fullness; therefore, it was apparent that the mass had been thoroughly collapsed and had remained collapsed. It perhaps was held in place by the pelvic organs. In a letter dated April 3, 1937, the patient said that she had returned to work and was feeling normal.

Comment

Spina bifida usually appears through defects in the lamina but it has been known to appear laterally through the spinal canal and in a few instances has appeared anterior to the vertebral column. I have had occasion to observe a previous case in which the spina bifida was situated anterior to the sacral vertebræ. There had been a projection of a diverticulum of the spina bifida through the rectum. The patient was operated on for a rectal polyp and death resulted. The case reported is unique in that the mass was situated anterior to the sacrum as a result of a congenital anomaly in the anterior portion of the sacrum. The tumor and the bony erosion resembled a dermoid situated in this position. The tumor also resembled a chordoma which had extended into the pelvis, except that a chordoma is much firmer to palpation than was the mass in this case. The digital examination of this particular mass gave one the impression of a distended bladder except that the mass was situated on the opposite wall of the pelvis. The case is also unique because a cure was effected without removing the pelvic sac. This further confirms my opinion that a spina bifida cystica is a herniation of membranes through a spinal defect and that a cure can be effected without bone graft and without a folding in of the membranes; all that was necessary in this particular case was a division and a ligation of the dural-arachnoid tube at a place where the caudal sac normally would end.

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CARCINOMA OF THE GALLBLADDER*

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IT is not with the idea of offering anything new that this paper is written, but to call attention to a condition which I think has not been given enough thought and consideration in the care of cases with gallbladder disease.

As the pendulum of surgical opinion swings from conservatism to radicalism, and back again, it is often difficult to establish the accepted course to pursue. In April, 1911, W. J. Mayo wrote, "Ten years ago, we heard a great deal about "innocent gallstones," which means that gallstones existed without symptoms and that their presence was not suspected until postmortem examination brought them to light. We can not now escape the conviction that gallstones did cause symptoms and that we, as diagnosticians, and not the gallstones, were innocent." Andrews would take serious exception to the above quotation, for he recently said, "I think the operation on silent stones is a scandal." The question I would raise at this time is, how often do we see "silent stones"? A careful history taken after we know that a certain patient has stones will usually elicit the fact that symptoms were present even though they were mild and entirely disregarded by the patient. It certainly is not necessary to have colics to have symptoms of gallbladder disease. As we have hypersensitive patients, we also have hyposensitive ones. The latter will uncomplainingly carry their troubles, and conceal symptoms which will be uncovered only by the most searching questioning.

Carcinoma of the gallbladder is of sufficient frequency to make it a factor of serious consideration when we must pass judgment on a case of gallstones. The exact percentage of malignancy is difficult to evaluate as many cases both of gallstones and malignancy go undiagnosed. It is usually ranked sixth in frequency of malignancy of the digestive organs. Surgical experience in various large clinics in this country place the percentage from 1 to 2.5 per cent of all operations on the gallbladder. Collected statistics from cases reported by Wilkie, Deaver, Smith, Judd and Gray, Miller, French, Sherrill and McCarty show that there were 393 carcinomas in 35,054 operations, an incidence of 1.12 per cent. Boyce and McFetridge[2] in the *International Surgical Digest*, Critique Section, state, "Looking at the facts from another angle a certain proportion of patients with gallstones develop cancer. Leutz, in 557 cases of cholelithiasis, found that malignancy developed in 5.1 per cent of women over thirty-nine years of age, and in 4.3 per cent of both sexes. Rolleston found the incidence of malignancy following cholelithiasis to be 4.5 per cent; Moynihan 5 per cent; Ridel 7 to 8 per cent; Graham 8.6 per cent and Schroeder 14 per cent. In five of the eighty-four cases reported by Magonn and Renshaw from the Mayo Clinic in 1921, the malignancy had developed at the site of a previous cholecystostomy for stones, and similar cases have been put on record by F. K. Smith, Mayo Robson, Knapp, Lett and others."[6] Judd has stated that those who develop malignancy of the gallbladder have had stones. Clinicians who will not accept this statement in its entirety must admit that cancer will not develop in a healthy gallbladder. An argument against this theory is the fact that experimentors have been unable to produce cancer in animals by the introduction of gallstones or other foreign bodies into the gallbladder. Barlow made the suggestion that calculi taken from malignant gallbladders are radio-active. Petrac and Krotkina used nineteen guinea pigs and inserted small radium tubes in twelve, and produced malignancy in two, after 136 and 158 weeks, respectively. However, two of the controls, in which only sterile glass capsules were inserted, also developed malignancy with metastasis at the end of sixty-six and 166 weeks. Other observers feel that a chemical factor either independent or in association with stones is responsible for the new growths. However, if we recognize the chronic irritation theory, there seems to be a causal relationship between stones and malignancy. As regards sex distribution, almost three-fourths of the reported cases have been in women, somewhat higher than the average distribution of benign lesions. There are records of carcinoma of the gallbladder in all decades, over the age of

*Read before the Saint Paul Surgical Society, February 10, 1938.

twenty, but the majority fall between the ages of fifty to sixty-five.

Papillomata are of such frequent occurrence that one would hardly believe that there is a relationship between them and malignancy. Nevertheless, cases have been observed where there are areas of epithelial proliferation extending through the muscle wall and some specimens show a close resemblance to papillary carcinoma. If there is at least a possible relation between these apparently benign looking, wart-like growths and the development of cancer, we then must look upon such a gallbladder as being better out, and not just regard it as many would, along with the strawberry type, as a metabolic disturbance incapable of causing any damage.

Pathology.—There are four generally recognized types of carcinoma of the gallbladder, and they can usually be differentiated by the naked eye: (1) scirrhous; (2) papillary; (3) mucoid-colloid; (4) squamous cell (epithelioma).

1. The scirrhous type is the most common, starting as a small infiltrating tumor, or thickening, which is at times overlooked in its early stage, but in its later development involves the whole gallbladder, contracting down on the stones so as to make it difficult at times to identify the organ itself. Microscopically, it is that of an adenocarcinoma It extends very early into the liver and the glands along the common duct.

2. The papillary type originates at the fundus or neck of the bladder and grows out into the lumen as a coarse villous or solid fungating mass. It is often associated with empyema as it early obliterates the cystic duct. Microscopically, it is that of a columnar cell adenocarcinoma.

3. The mucoid, or colloid type, is bulky and soft, and can be recognized by its jelly-like appearance. Microscopically, there are trabaculæ of fibrous tissue with large spaces of pseudomucin. Tumor cells are few and are found singly or in small groups. These cells contain small drops of mucin which enlarge, bursting and discharging their contents into the connective tissue.

4. The squamous type is the least common, and can as a rule only be differentiated microscopically. It is seen as an epithelioma made up of cells of squamous type with prickle cells, and only occasionally do epithelial pearls occur. The usual explanation of a squamous cell type in the gallbladder is that it arises from a previous leukoplacia which has been caused by irritation from pre-existing gallbladder disease.

Dissemination of any type is usually by local excision, distant metastases being encountered at post mortem. The lungs are involved in 10 per cent of the cases. The liver, on account of its intimate association and direct blood and lymphatic supply, is involved very early. In the right lobe, there frequently forms a large hard mass, while, at times, multiple nodules are found throughout the entire organ, which becomes greatly enlarged. The regional lymph nodes also are the seat of early metastases. The node by the cystic duct enlarges, blocking both the duct and artery, resulting at times in hydrops or more frequently empyema. The carcinomatous bladder is more subject to empyema than others. From this first gland, the others along the common duct and retroperitoneally become invaded.

Clinical Features.—Apparently, as yet, we have no signs by which an exact diagnosis of carcinoma of the gallbladder can be made in its early stage. The usual symptoms of gallbladder disease are present in the well taken history, varying in degree from those of a severe colic to the mildest indigestion. In two recently observed cases, there was a distinct change in the character and degree of the distress, the discomfort becoming more continuous and unrelated to food intake. The colic present earlier in the course of the disease in these cases had given way to more constant pain, which was the one symptom that finally made the patients consent to operation. Nausea, vomiting, anorexia, weight loss and jaundice, and finally cachexia, with or without signs of emypema of the gallbladder, are symptoms which appear as the disease advances. Jaundice may be an early symptom if the glands become involved and cause pressure on the common duct. The earlier the jaundice occurs, the sooner death will follow. Diarrhea and extreme weight loss have been noted in certain cases before any symptoms of biliary disease have attracted attention.

Various authors have tried to classify the disease according to symptoms but the overlapping and bizarre complaints make this extremely difficult. If we wait until a tumor mass is felt before advising operation, we will find that the patient is well beyond any surgical relief. This is the stage in which most of the patients are seen.

Cholecystography might be of possible value in early diagnosis, as Kirklin has suggested that a filling defect more than 2 cm. in diameter with an irregular border should arouse suspicion of malignancy. The necessity of well taken films and proper interpretation, of course, is very obvious. Boyce and McFetridge further observed in this review: "The only solution of the problem seems to be the constant recollection that malignancy of the gallbladder is a definite possibility in all cases of persistent retention of bile and the consideration of malignancy as a possible diagnosis of all cases of cholecystitis or cholelithiasis in individuals over forty, or even younger. If the possibility of its occurrence is not constantly borne in mind, the diagnosis will always be missed."

Treatment.—At the present time, the majority of patients with carcinoma of the gallbladder are seen too late for radical removal. Any operation, except in the very early cases, usually renders the patient more uncomfortable than before. If a cholecystectomy is at all feasible, it should be done and the lymph nodes along the cystic duct removed. The electrosurgical unit is of value for this operation as a portion of the liver about the gallbladder fossa where direct extension takes place should be removed. Gastroenterostomy may, at times, be indicated to relieve the pyloric obstruction. The immediate mortality, especially in the jaundiced cases, is usually very high, and those that survive operation, succumb in a few months. Every series on record has the same discouraging picture. Webber, at the Mayo Clinic, studied thirty cases according to Broders index, correlating the clinical findings with the microscopic picture. He found that in the groups graded I and II, palpable tumors were present in two cases, while there were twelve in Grades III and IV. Furthermore, the duration of life in Grades I and II was thirty-four months against 4.8 months for those of Groups III and IV.

Early operation in gall bladder disease will, undoubtedly, prevent the occurrence of carcinoma, as well as ward off many of the other possible complications. Heyd, former president of the American Medical Association, writes editorially, "Preventative medical thought and wise judicious surgery would suggest the early removal of chronically infected gallbladder and not delay until the accident of infection initiates a fulminating acute cholecystitis," and further he states, "Teachers of surgery who lend their prestige and give support to a policy of waiting, provide authority for timid surgeons, inexperienced operators and procrastinating practitioners."

Having personally observed patients with gallstones go through long periods of watchful waiting, hoping that nothing very serious will develop except an occasional colic, and then later seeing them with the more serious complications of damage to liver and pancreas, empyema, and, in a few, malignancy, I can wholeheartedly subscribe to Dr. Mayo's saying: "Innocent gallstones —a myth!" I believe the mortality in gallbladder operations performed before the development of complications should, in competent hands, be less than the incidence of malignancy alone. Individual case reports are of very little value as the usual picture is well known, but upon seeing a small but increasing number of these cases, which might have been avoided but for procrastination on the part of either patients or their physicians, I feel that too little attention has been called to this usually avoidable tragedy.

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RECURRENT "TROPICAL" LYMPHANGITIS*

With Report of a Case

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SIMPLE acute lymphangitis is a rather common affection accompanying infected wounds of the hands and feet, and is usually due to infection from streptococci, less often to staphylococci, gonococci, or pneumococci. The bacteria may enter the lymph vessel directly from the infected wound, or pass through the wall of the vessel from without. However, inflammation of the lymphatics may occur without a primary focus of infection. It may occur during the course of acute infectious diseases, herpes, and especially erysipelas. Such chronic infectious diseases as gonorrhea, syphilis, tuberculosis and bubonic plague may show lymphangitis. Other forms occur after roentgen irradiation, sunburn, poison ivy and insect bites.

Osler[5] offers a pathologic classification of acute lymphangitis as follows: simple, purulent, and proliferative. The chief features of the simple form are hyperemia, edema and infiltration of the vessel walls and perilymphatic tissue with a resulting thickening which may become necrotic or proliferative. The condition returns to normal if the existing cause is removed. Otherwise, it may lead to a chronic process.

The purulent form shows more thickening of the wall, the lumen becomes filled with pus or a fibrino-purulent mass which, between the valves, gives a beaded appearance. Abscesses may form along the course of the lymph vessel or in the regional lymph nodes, and septicopyemia may result.

The acute proliferative type occurs chiefly in gonorrhea, the principal feature being a marked perilymphangitis.

Chronic or recurrent lymphangitis results in a partial or complete obliteration of the lymph vessel due to hypertrophic changes resulting from a proliferation and induration of the connective tissue of the vessel wall and surrounding tissues. When affecting an extremity, chronic edema or elephantiasis are the most important clinicial features.

Goeckerman,[1] in discussing recurrent lymphangitis, distinguishes between lymph-edema and solid edema, applying the former term to stasis of lymph resulting from mechanical obstruction like that following radical amputation of the breast without infection, while restricting the term solid edema to the end-process of recurrent lymphangitis.

Recurrent lymphangitis may be defined as repeated attacks of simple acute lymphangitis, due to an inflammatory process in the cutaneous lymphatic vessels caused by the streptococcus hemolyticus. The involved part shows a well defined area of redness, swelling and local pain, which spreads by direct continuity and is associated with general febrile symptoms. Suppuration may occur when the subcutaneous tissues are involved, and the whole process turns into the suppurative form. It occurs in temperate climates during the hot months, but is much more common in the tropics or sub-tropics, where it is called recurrent tropical lymphangitis. This is probably due to the fact that profuse sweating, dehydration and irritation of the skin predisposes it to infection. Although erysipelas affects the skin somewhat similarly it is more frequent during the colder months of the year, probably due to the increased frequency of upper respiratory infections. Also meteorological studies have shown that the frequency of erysipelas corresponds with an increase of humidity in the air. Other streptococcic infections, such as scarlet fever and rheumatic heart diseases, are common in temperate and cold climates, but scarce in California, and almost unknown in Puerto Rico.

My case is that of a white male, thirty-one years of age, single, who for the past eleven years has had recurrent attacks of inflammation of the lymphatics in the lower extremities, followed by edema.

He was born in Kentucky in 1906 and lived there for four years, before moving to Ohio. During early childhood he suffered no other illnesses than an attack of typhoid fever at the age of two and a second attack at the age

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of eleven. At twelve he was seriously ill with an acute bronchitis and an associated jaundice which persisted for a period of five weeks. A tonsillectomy was performed at the age of sixteen. There was no history of venereal diseases. His father and mother are living and well. His grandmother, mother, and brother have had acute rheumatic attacks, accompanied by high fever, and a moderate amount of swelling and pain in the hands and feet, lasting about a week.

The patient was apparently in good health until August of 1925, at which time he had a blister on his left heel which became secondarily infected. This condition cleared up in about a week under treatment, but recurred a month later and necessitated hospitalization for a ten day period. At the end of that time there was no further evidence of infection and he remained in good health until August, 1926, when he sustained an abrasion to the skin of the lower left leg. Within twelve hours he had a severe chill followed by a high fever and the typical clinical picture of an acute lymphangitis and lymphadenitis of the left leg and groin. The treatment consisted chiefly of rest, elevation of the limb, and application of local wet packs. The infection gradually subsided and within five weeks he was able to return to work.

In July, 1927, during a spell of very hot weather, without any evidence of external injury, he developed pain in both groins with tender, swollen glands, and evidence of lymphangitis of both legs, accompanied by high fever and the same constitutional symptoms as before. This attack subsided in ten days, after which he resumed his work.

Similar attacks occurred the next three summers, always during spells of hot weather, with complete recovery from each attack, except that there was a persistent gradual increase in the edema of both lower legs. He had no recurrence during the summer of 1931, all of which time he spent in Duluth, Minnesota. From 1931 to 1936 he had somewhat milder recurrences each summer, the most severe attack occurring in August, 1936, at which time he first came under my observation. About two weeks before this attack he came to my office complaining of edema of both lower legs below the knees.

Physical examination at that time was entirely negative except for edema of the lower extremities and an epidermophytosis infection between the toes, commonly known as "athlete's foot." Tests for patency of the internal veins of both legs proved them to be normal. Urinalysis showed an occasional cast; P.S.P. 77 per cent in two hours; hemoglobin 83 per cent; rbc. 4,170,000, wbc. 8600, P.M.N. Neut. 69 per cent, P.M.N. Eos. 4 per cent, large mono. 1 per cent, large and small lympho. 26 per cent; blood urea 15.6 mg. per 100 c.c. blood; Wassermann negative; basal metabolic rate minus 3.

A sedimentation rate of 55 mm. in 45 minutes was the only finding of consequence in any of the blood tests.

Six days later, during a spell of very hot weather, he suddenly had a severe chill, followed by a temperature of 105, and became acutely ill. Both lower legs were swollen, rather tender, with glandular enlargement of both inguinal regions. These glands were acutely inflamed, tender to touch, and they were more numerous and larger on the left side. He was sent to the Swedish Hospital, where laboratory tests revealed the usual febrile urinary findings, white blood count 30,500; differential count of 91 per cent P.M.N.; 5 per cent large Mono., 3 per cent small Mono., and 1 per cent transitional cells. The blood culture, Wassermann and tests for tularemia mellitensis and typhoid were negative.

The patient's temperature was 104.2 degrees on admission to the hospital and increased to 105.6 shortly afterward. He was irrational at this time. A septic temperature curve persisted for the next five days, fluctuating between 100 and 103 degrees, and gradually descending to normal on the sixth day.

The most interesting finding was the appearance of the extremities. There was an area of redness and a rash beginning at the ankle and spreading upward. The skin became hot, swollen and glistening; the margin of the rash was sharply demarcated but not palpably raised. As the rash advanced, the center of the affected area became somewhat pale and edematous. It spread from below upward by direct extension, and extended to the knee joints or slightly above. However, a red streak extended up along the cords of the lymphatics to the regional lymphatic glands in the groins. As the temperature began to fall and the general con-

dition of the patient improved, the rash concurrently began to fade and practically disappeared when the temperature was normal. However, the limbs remained swollen and painful for many days afterward, and there was slight desquamation of the skin on the affected parts. The edema persisted and continued to remain increased over that present before the attack.

The above description is typical of all his attacks, but some, however, were not as severe as this one. Except for the persistent edema the patient remained well until August, 1937, when, during a spell of very hot weather, he had a similar but milder attack while working in North Dakota. This attack lasted about eight days and then left him again with a slight increase in the edema of the extremities. On close quizzing I found he had noted peeling between the toes practically every summer, and had treated this during one previous summer, following an attack. Altogether the patient has had fifteen attacks. Several summers he has had two attacks, and in 1930 had three attacks. The only summer he was free from it was in 1931 when he spent the entire summer in cool Duluth, Minnesota. In 1929 he received vaccine from a gland removed from the groin by Dr. Duff, but apparently this was of no help. The microscopic examination of this gland showed only chronic inflammation. He suffered no complications in any of the attacks except a persistent pachyderma.

I feel that my case is similar to those reported by Janero Suarez[7] under the title "Recurrent Tropical Lymphangitis." This disease occurs quite frequently in the southern part of the United States and is endemic in Puerto Rico and other sub-tropical countries. It attacks chiefly young people, more commonly females, who are most susceptible during menstruation and the puerperium. Over 90 per cent of Suarez's series occurred in the lower extremities, 81 per cent being limited to one leg. When present in both legs, one is usually more affected than the other. It gives no immunity to the patient; on the contrary, one attack predisposes to another. Recurrences may take place frequently or may be months or years apart, and are most common between the ages of twenty and forty. However, it has been noted that a severe attack is usually followed by a long remission. Patients have been known to have sustained fifty or sixty attacks, often occurring regularly, every four to six weeks. Chronic edema or pachyderma results in a majority of the cases. Suarez believes the mechanical lymphatic obstruction or bacterial infection is in itself capable of producing elephantiasis. Mates[4] and Grace[2] believe that bacterial infection plays an essential etiological role in the pathology of elephantiasis. Drinker[3] and his associates have demonstrated in remarkable experiments on large police dogs that spontaneous attacks of lymphangitis occurred where lymphedema or experimental elephantiasis was produced by total destruction of the lymphatics in the hind legs. Hemolytic streptococci were cultured from the tissues of the affected leg, which could be transferred to other dogs, but only in limbs previously operated upon. In view of the fact that humans are more susceptible to streptococcal disease than canines, previous obstruction of the lymphatics is not essential for the production of acute lymphangitis, but, when present, predisposes to future attacks.

In Suarez's[7] series of 139 cases, ninety-one showed a definite focus of infection, 72 per cent of which were epidermophytosis interdigitalis. Removal of this infection was usually followed by cessation of attacks. One can usually get a history of focal infection or injury preceding the first attack. Occasionally cases occur in which no local lesion can be found. Suarez had two cases of this kind. They were classified as allergic. The redness, heat, edema and fibrosis probably represent "an allergic phenomenon to a protein fraction of the streptococcus or its toxin."

The symptoms and physical findings during a typical attack in my case correspond well with those described in Suarez's series. Incubation period was two to three days, while relapses occurred in 4 per cent of his cases. Chronic lymphedema and subcutaneous abscesses are the most common complications.

Otero and Lebron[6] demonstrated the presence of agglutinins against streptococci in practically every case of recurrent tropical lymphangitis. They give a positive allergic reaction to streptococcus filtrate, which disappears during the febrile stage. However, they found a definite increase in the antistreptolysin content of the blood, during an attack. They also believe that

acute attacks are preceded by hemolytic streptococcus infections.

The presence of an area of redness with a well-defined margin, spread by direct continuity, pain and the accompaniment of constitutional symptoms, distinguishes recurrent lymphangitis from other erythematous lesions. Abscesses, cellulitis and thrombophlebitis may at times be confused.

Prognosis is very favorable, no deaths having been reported. Morbidity is great, however, due to recurring attacks. Disability produced by the elephantiasis is of considerable importance.

Treatment consists of the following:

1. Removal of any possible focus of infection.
2. Proper care of the feet, especially during hot weather, with elimination of any possible epidermophyton infection.
3. Goeckerman[2] has had gratifying results in the early stages in seventeen cases from the use of foreign protein given intravenously and filtered roentgen rays applied locally.
4. Moving to a cool climate during the hot summer months is good prophylactic treatment.
5. The treatment of the elephantiasis is a difficult and lengthy problem.

Conclusions

1. I believe this case represents a definite clinical entity, occurring sporadically in this country, more commonly in the south, but which is endemic in tropical countries and described under the title of recurrent tropical lymphangitis.
2. It occurs only in hot weather.
3. An infective lesion or abrasion can usually be found, especially in the initial or early attacks. The initial attack predisposes the patient to further attacks. Severe attacks may produce some temporary immunity.
4. Injury to the lymphatics of the extremity with resulting chronic obstruction and an acquired allergy to hemolytic streptococcus, plus hot weather, may cause recurrences without infection or injury to the skin. This has been borne out by clinical and experimental observations.
5. Treatment, except prophylactic, has been unsatisfactory.

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INTESTINAL OBSTRUCTION DUE TO CALCIFIED MESENTERIC GLANDS

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IN ROUTINE roentgenograms of the abdomen shadows of calcified mesenteric glands are a rather common finding. In the large majority of cases they are more or less of academic interest and surgical interference is not indicated. It is interesting to speculate upon their etiology and pathogenesis.

The primary causative agent is of course the tubercle bacillus. The human and the bovine types have been isolated from the glands. The human type is isolated from the glands of patients with tuberculosis elsewhere in the body whereas the bovine type is isolated usually from the glands of children who have ingested milk from tuberculous cows. Approximately 60 per cent of the cases are of the latter type.

The clinical course of the disease is variable. In the majority of patients no definite history of symptoms which might lead one to suspect the disease can be elicited. The disease is discovered late when a roentgenogram of the abdomen is made. The roentgenographic findings, when the diagnosis is made this way, are typical shadows of calcified glands usually having characteristic features. They present moth-eaten shadows of different density varying in diameter

Fig. 1. Roentgenogram showing a large mesenteric gland in the right lower quadrant of the abdomen, measuring 8 x 5 centimeters. Gaseous distention of the small bowel can also be seen.

Fig. 2. Roentgenogram showing a small mesenteric gland in the right lower quadrant of the abdomen measuring 2 x 2 centimeters. The degree of obstruction may be demonstrated by the dilated loops of small bowel.

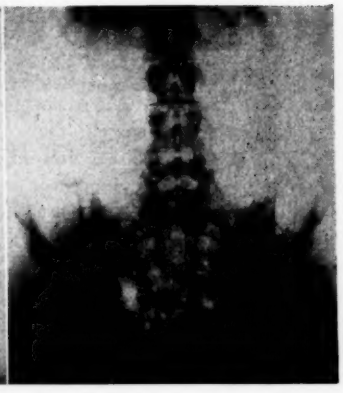

Fig. 3. Roentgenogram showing two large and multiple small shadows of calcified mesenteric glands. The largest measures 4 x 3 centimeters. No dilatation of the bowel is evident.

from one to five or six centimeters or more. They may be found any place in the abdomen but are usually located in the region of the umbilicus, probably a little more often on the right side.

Early in the disease an entirely different picture may be seen. The so-called acute type may give rise to definite clinical symptoms and signs. These are usually pain, colicky in character, associated with nausea and vomiting and abdominal tenderness in the region of the umbilicus or over the right lower quadrant of the abdomen. Exploration at this time will reveal enlarged caseating nodes in the mesentery.

Subacute types may be characterized by indefinite and vague abdominal complaints associated with pallor, weakness, and occasional attacks of slight fever. At times, the glands may be palpated.

Recently, we have observed three cases which were of the asymptomatic chronic type until obstruction of the bowel developed. From the size of the glands found at operation and because of their hard calcified nature, it was apparent that they had been present for many years, but the patients had had no symptoms referable to them. In two of the three patients, complete obstruction of the bowel was produced in the first attack, and in one chronic recurring partial obstruction was produced. The diagnoses were made from the findings of obstruction and the presence of shadows in the roentgenograms suggestive of calcified mesenteric glands.

Case 1.—A seventy-one-year-old woman was seized by severe abdominal cramps and abdominal distention. Within twelve hours vomiting occurred. This persisted. The patient was unable to defecate or pass flatus even with enemata. A roentgenogram (Fig. 1) revealed a large calcified shadow suggestive of a mesenteric gland. At operation through a right rectus incision, the entire small bowel was found injected and distended. The point of obstruction was in the right lower quadrant involving the ileum about twelve inches above the ileocecal valve. A large calcified gland was present in the mesentery close to the bowel. Cicatrization formed adjacent to it completely obstructed the bowel. The gland was excised, a few adhesions cut, and the collapsed bowel distended as the gas passed on. The patient made an uneventful recovery.

Three months later, she again presented herself with obstruction of the bowel. This time, it was due to a strangulated right inguinal hernia. This was freed and the hernia repaired. She again had an uneventful convalescence.

Case 2.—A forty-six-year-old married woman was awakened at two o'clock in the morning by severe abdominal cramps. She slept very little during the remainder of the night. A hypodermic injection of morphine was administered at nine a. m. The patient was first seen at eight p. m. Paroxysms of pain were present and visible peristalsis could be seen. She obtained slight relief from enemata. A roentgenogram of the abdomen (Fig. 2) showed distention of the bowel and a shadow suggestive of a calcified mesenteric gland. Exploration, through a right rectus incision, revealed a greatly distended upper ileum. An obstructing band at the site of a calcified mesenteric gland encircled the bowel. This was cut and gas escaped into the collapsed bowel beyond the obstruction. The gland was excised. The patient made an uneventful convalescence.

Case 3.—A twenty-five-year-old single woman had had an appendectomy five years previous to the time I saw her. She complained of intermittent attacks of abdominal pain associated with abdominal distention. This pain could be relieved by enemata. Roentgenograms of the abdomen revealed two shadows of calcified mesenteric glands (Fig. 3). At operation, a constricting band bound the bowel to one of the glands. This was cut and the glands excised. Convalescence was uneventful and there has been no return of pain.

BROMIDES, THEIR USE AND ABUSE*

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WITH the gradual elimination of industrial hazards, with increased efficiency in the control of epidemic diseases, and with the speeding up of modern living conditions, there has been in recent years a great increase in the number of functional nervous diseases afflicting the general population of our country. Man formerly used his skeletal muscles to subdue his natural enemies and to convert natural resources into the essentials of life. In modern times the burden has been lifted from our physical bodies and a great load thrown upon our psychological and emotional mechanisms. As a result of this change, the administration of hypnotics and sedatives which act upon the nervous system represents one of the most common procedures in modern medical practice. Millions of dollars are spent annually for the purchase of these drugs, and as a result of carelessness on the part of the medical profession they are frequently used by the laity without medical supervision. These drugs effect no cure. Their function is merely to mitigate; to intercede between man and his environment; to serve as a buffer which absorbs part of the constant and unrelenting barrage of stimuli to which man is subjected. There is scarcely any group of drugs more freely employed than sedatives and hypnotics, and prominent in this group are the salts of bromine. Inasmuch as the functional conditions in which these drugs are given are usually prolonged states, cumulation often occurs; but because of their extreme diversity, the symptoms of bromide intoxication are frequently missed. The complex of the busy doctor, plus the neurotic patient, plus the "salty medicine," is a common one; and the oft-repeated statement, usually delivered over the telephone, "keep taking that salty medicine," many times does much more harm than good. There are times when I suspect that a physician's skill as a psychotherapist stands in inverse proportion to the amount of bromide that he prescribes.

True, the salts of bromine are too valuable as therapeutic agents for us to decry their use, and it would be no more logical merely on account of a few cases of bromide poisoning to condemn them as a whole than it would be to condemn digitalis because of the occasional occurrence of digitalis poisoning. In my experience, however, bromide intoxication seems to be occurring with such increasing frequency that, in spite of numerous contributions already made to the literature on the subject, it seems justifiable again to call the attention of physicians to certain dangers inherent in the use of bromides, and to point out specific methods for the diagnosis and treatment of bromide intoxication. Credit should be given to Wuth[17] for awakening interest in bromide intoxication and for publishing in 1927 a simple laboratory method for the quantitative estimation of the bromide content of blood serum.

The purpose of this paper is to call attention to the fact that bromides even in therapeutic doses may produce mental symptoms in susceptible individuals. These symptoms range from a mild clouding of consciousness to an active delirium, and from mental depression to stupor and coma. In recent years, I have observed a number of patients who have manifested symptoms of bromide intoxication which have slowly cleared up after withdrawal of the drug. These have usually been elderly individuals with varying degrees of arteriosclerosis, or cardiac and renal insufficiency; or younger persons who could not or would not take sufficient food, or were anemic and undernourished. It is probable that some of the exacerbations of mental symptoms and of the psychoses which occur from time to time in patients under our care are due to drug intoxication from our therapeutic efforts rather than to the underlying conditions which we are trying to treat. One has frequently seen acutely disturbed psychotics in full restraints and requiring feeding by nasal tube become quiet and coöperative within forty-eight hours after discontinuance of a bromide ration of 45 gr. (3.0 gm.) a day.

Incidence

The first case of bromide intoxication was reported by Huette[8] in 1850, and for many years following this, reports of the condition were very infrequent. With the shift in preponderance

*Thesis presented before the Minnesota Academy of Medicine, October 13, 1937.

from infectious and physical diseases to neuropsychiatric disorders, the use of the salts of bromine as therapeutic agents has increased and more and more cases have been reported in recent medical literature. Apparently, the incidence of bromide intoxication and bromide delirium is higher than one ordinarily suspects. Among 505 admissions to the state hospital at Harrisburg, Pa., during the two years beginning June 1, 1931,[6] there were fifteen unquestionable cases of bromide psychosis. Among 238 admissions to Johns Hopkins hospital over a six month period[17] there were twenty cases of bromide intoxication, an incidence of 8.4 per cent. Among 1,000 consecutive patients admitted to the Colorado psychopathic hospital, forty-four showed an excess of bromide in the blood, and in seventeen (1.7 per cent) the mental symptoms were due solely to bromism.[13] During 1935, 2.7 per cent of the patients admitted to the Psychiatric Clinic at the New Haven Hospital in Connecticut showed definite signs of bromide intoxication.[7]

Pharmacology[14]

The effects of the bromine ion are largely confined to the central nervous system. Whatever other effects it produces are relatively insignificant and may be considered as secondary to those on the central nervous system. The slowing of respiration, the reduction of body temperature, and the slowing of all the physical processes may be attributed to lessened movement and consequent decreased heat production incident to the depressing action upon the central nervous system. All parts of the nervous system are affected, but the higher cortical centers seem to be most susceptible to the drug. Thomas[11] working with rabbits found that the amount of bromide absorbed by various portions of the central nervous system depends, in part, upon the water content of its various tissues. By feeding different rabbits known quantities of bromide, Thomas was able to show that the bromide content of the gray matter of the central nervous system was about 150 per cent that of the white matter when the content was calculated in milli-equivalents of bromide per gram of dry weight. Bastido[1] has shown that in bromidized dogs stimulation of the motor areas does not produce convulsions. In humans under the influence of bromide, the higher intellectual centers are retarded and the reaction time is prolonged. There also is a depressing effect on the spinal cord and the spinal reflexes are inhibited. Ordinary doses of bromide have no effect on the circulation, but larger doses depress the heart and vasoconstrictor centers, lessening arterial tension.[9]

When ingested, bromides are readily absorbed from the stomach and appear in the urine a few minutes later. However, elimination takes place very slowly, and after a single does of bromide by mouth, although the drug may be detected in the urine in a few minutes, it also may still be found in the urine as long as twenty days afterward. Bearing this in mind one should not have any difficulty in appreciating the cumulative effect of oft repeated doses of bromide over a long period of time.

Landheimer, in 1901, was the first to recognize the important influence of the intake of chlorides upon the retention of bromides. Bromides are not excreted by the kidneys as rapidly as chlorides but they tend to displace the latter in the blood and other body fluids. The displaced chlorides are excreted by the kidneys in preference to the bromides. This permits an accumulation of the bromides with consequent lowering of the chloride content of the body. A diminished chloride intake (insufficient food, or a nasal feeding formula containing insufficient chloride, *vide infra*) increases the risk of bromide intoxication. Conversely, an increased chloride intake facilitates the elimination of bromide. When 25 to 30 per cent of the blood chloride is replaced by bromide, symptoms of intoxication usually appear. Bennoulli states that a replacement of more than 40 per cent of the chlorides in the blood by bromide is fatal. It can be seen, therefore, that when a patient is receiving bromide medication, his chloride intake is of paramount importance. In order to maintain the chloride-bromide equilibrium, according to Rylan,[8] the chloride intake should be four times the bromide intake. If the bromide intake equals the chloride intake, intoxication will occur in about three weeks. According to Wuth[17] the average hospital patient takes about 7.4 gm. chloride in twenty-four hours. At Mounds Park Sanitarium, in St. Paul, the average daily diet contains 12 gm. of chloride exclusive of what the patient adds to his food after it is served to him. This amount of chloride is enough to balance 3 gm. (45 grains) of bromide per twenty-four hours, provided the

patient eats all of his food. However, the standard nasal feedings, for which there are several formulæ, contain an average of 3 gm. of chloride per twenty-four-hour ration.[13] The average twenty-four-hour bromide dose for a neuropsychiatric patient being 3 gm. (15 gr. three times a day) it can readily be seen that if Rylan's computations are correct, patients on nasal feeding and regular bromide medication should show signs of bromide intoxication in about three weeks. In my experience this has frequently been the case.

The question has frequently been raised as to whether the effect of bromide is dependent primarily upon the excess of bromide itself or upon consequent diminution of the chlorides. The effect of a single dose of bromide is too rapid for chloride diminution to be a factor. Furthermore, it is possible to narcotize dogs with bromide before diminution of chlorides has had time to play any conspicuous part.[14]

Symptoms of Bromide Intoxication

The clinical picture of bromide intoxication is extremely variable, and according to Katzenelbogen[14] and his associates, the only accurate method of establishing the diagnosis is the determination of an excess amount of bromide in the blood. While it is true that there are no pathognomonic symptoms of bromide delirium, and while it is also true that one encounters many cases in which the cutaneous manifestations of brominism are absent, I believe that it is possible to separate the types of brominism into two large clinical groups: simple bromide intoxication or depression, and bromide delirium or psychosis.

Simple bromide intoxication or depression consists of a state of mental sluggishness in which the patient thinks and acts with great difficulty, but is oriented and doesn't exhibit delusions or hallucinations. He is dull, stupid, and indifferent. His face is pale, expressionless, and may or may not show a rash. The eyes are heavy lidded, the expression staring, and the patient complains of headache. All the mental and physical reactions are retarded, memory may be impaired for recent events, and the gait is ataxic. There is anorexia, flatulence, nausea, and constipation. All tendon reflexes, as well as the corneal and pharyngeal reflexes, are reduced or absent. The pulse may be rapid and the temperature somewhat elevated.

Bromide delirium or psychosis, as has been stated before, is not an independent nosological entity. It is like any other toxic delirium and belongs to what Adolph Meyer calls the dysergasic state. The dysergasic state embraces all the exogenous psychoses, acute delirious manias, toxic-infectious psychoses, infective-exhaustive psychoses, deliria of fever, and drug deliria described by other authors. In addition to disintegration of function at the biological level (ataxia, tremor, slurring speech, et cetera) there is always dysfunction at the psycho-biologic level. Consciousness is clouded to a degree varying from patient to patient, and often from one minute to the next in the same person. There almost always is disorientation to some degree in all spheres, and the outstanding feature is great distractability. Thinking is incoherent, and there may be delusions and hallucinations which run a rapid course. The hallucinated objects are usually moving and multiple. Sometimes there are illusions in which the patient's bed becomes a boat or a train and his conversation indicates that he has a sensation of being in motion (vestibular irritation?). Some patients have transient manifestations of the Korsakow phenomena. Restlessness may become extreme and the patient may call out loudly, shout or scream, and become very disturbing to others. Restraints usually are necessary because patients react to hallucinatory experiences and delusional ideas. They try to join relatives whom they fancy are in an adjacent room, they may run away, jump out the window, et cetera. There sometimes is miming of familiar or occupational activities, and the miming may be of varying degrees of elaboration such as sewing, smoking or going through the complicated movements of dressing or undressing. Wolff and Curran[16] cite a bromide patient who had a police record and fancied that he was in a bootlegging drama. He thought that he was about to be "put on the spot" and enacted a scene rich in activities of fear and defense. Another patient built a bathing platform and jumped from the top of the table to the floor which he believed to be a lake.

One frequently must answer the question: "In a given patient, for what part of the psychotic picture are the bromides responsible?" Levin[5] states that if in an attempt to control the symptoms of a psychosis a patient is given bromide and there is no subsequent change in the psychotic picture, the case cannot be regarded as

one of bromide psychosis. On the other hand, if following the administration of bromide the psychotic picture changes, the possibility of a bromide delirium must be considered. Bromide deliria usually clear up in from two to six weeks following discontinuance of the offending drug. However, either the underlying psychosis may persist after termination of the delirium and cause the psychiatrist mistakenly to dismiss from his mind the possibility that a bromide delirium ever existed, or a bromide intoxication theoretically may give rise to a chronic psychosis so that a patient who is not psychotic before the delirium fails to return to his normal health many months after the bromides have been discontinued.

Harding and Harding[2] believe that there exists a rough correlation between the amount of bromide in the blood and the type of mental reaction resulting therefrom. Patients with simple bromide intoxication or depression usually show a serum bromide of 100-150 mg. per 100 c.c. while a serum bromide of 150-300 mg. produces bromide delirium or psychosis. Serum bromides above 300 mg. c.c. often cause the patient to become more or less stuporous. On the other hand, Katzenelbogen, et al,[4] studied a number of cases and concluded that there was no distinct correlation between toxic symptoms and the bromide level in the blood. Patients with serum bromide as high as 385 mg. did not show any clinical symptoms of bromide intoxication; in other cases, acne, loss of pharyngeal reflexes, vertigo, ataxia and weakness were observed in patients with serum bromide as low as 147 mg. These workers conclude, therefore, that in following patients under bromide treatment it is the clinical picture and not the laboratory findings which should be primarily reckoned with in regard to the menace of bromide intoxication. According to Wuth,[17] however, any serum bromide in excess of 150 mg. should be considered as being in the "toxic zone." Max Levin[6] agrees that a serum bromide over 150 mg. will tend to produce symptoms. Moreover, when the serum bromide is high, the psychosis may begin to clear up before the content gets back to 150 mg., or it may persist after it has fallen below the level. The underlying psychiatric makeup of the patient has a great deal to do with this. Walter[15] suggests that it might be due to altered permeability of the meninges.

Course

The duration of symptoms of bromidism following discontinuance of the drug is from two to six weeks. According to Levin[5] this duration is determined by the following factors:

1. Organic brain disease which undoubtedly retards recovery.
2. Pre-existing psychoses which probably have the same effect.
3. Even in the absence of 1 and 2, there probably are constitutional differences in the speed with which delirium clears up.
4. The duration after discontinuance may vary according to the duration of symptoms before discontinuance of the drug. For example, a delirium which had been present for several weeks before the bromide was discontinued probably will take longer to clear than one which has lasted for only a few days.
5. The administration of sodium chloride probably hastens the clearing up of the delirium.

Treatment

Naturally, the first thing to be mentioned under the treatment of bromide intoxication is prevention. Bromide dosage should be kept down to the minimum and should not be continued over a long period of time. A patient receiving bromides must receive chlorides. One must be especially careful in dealing with old people, arteriosclerotics, and patients suffering from cardiac decompensation or renal insufficiency.

When bromide intoxication does occur, the first thing to do is to discontinue administration of the drug in spite of intense restlessness and a disturbed mental condition. Bromides, as well as all other sedatives, must be withheld. Some writers believed that there was a "withdrawal delirium" probably analogous to the psychotic manifestations accompanying the withdrawal symptoms of chronic morphinism. However, in the case of bromides, the delirium depends upon a certain concentration of bromide in the blood, together with the resistance of the patient. As far as is known, following the abrupt withdrawal of bromides, there are no secondary reactions unless something intervenes to reduce the resistance of the patient. For this reason, the first procedure in the treatment of brominism should be to discontinue the drug. Next, the chloride content of the blood must be increased and this is best done by the intravenous administration of

normal salt solution and the giving of Na Cl by mouth. I usually give a capsule of 15 gr. (1.0 gm.) Na Cl four times daily. Patients who are very restless usually respond to hydrotherapy in the form of continuous baths or neutral body packs. When giving packs, care should be exercised that the pack is not warm enough to cause the patient to perspire excessively, for in that manner more chloride will be lost from the body. Elimination through the intestinal tract may be accomplished by a course of calomel followed by a saline cathartic. Sometimes spinal drainage helps to overcome restlessness and to eliminate bromide from the central nervous system.

With patients who are extremely disturbed and who are approaching a state of exhaustion and in whom the need for immediate rest is imperative, either paraldehyde by mouth or sodium amytal intravenously is usually adequate. I have used retention enemas of paraldehyde in olive oil, but with this form of administration there is some danger of producing gangrenous proctitis. Additional treatment of bromide intoxication or delirium is purely symptomatic and not complicated.

Toenhart[12] states that with bromides in the blood serum, hydrobromic acid is secreted by the stomach, neutralized, absorbed by the intestine, and later re-excreted into the stomach, thus producing a vicious circle. If in bromide intoxication the ratio of bromides to chloride is anywhere near the ratio of these ions to each other in the blood stream, then gastric aspiration should offer a rational and effective method of relief. Toenhart advocates gastric aspiration repeated several times daily over a period of several days. This removes as much of the bromide as possible, but chloride is necessarily removed also, so must be replaced either intravenously or orally. With Toenhart's method it is possible to remove as high as 1,240 mg. (20 gr.) of bromide in eight hours using continuous gastric aspiration with a negative pressure apparatus.

Many cases diagnosed as bromide delirium or psychosis were seen in hospitals not equipped to run serum bromide determinations, so we cannot consider these as proven cases. However, the history, clinical picture and subsequent course in every one of the unproven cases is so characteristic that we seem justified in assuming that they were cases of true bromide psychosis. Some of these were superimposed upon pre-existing psychoneurotic conditions. Only a few typical ones will be cited, and then we will go on to proven cases. For the sake of brevity irrelevant features of the histories, physical findings, and clinical courses will be omitted.

Case Histories

Case 1.—A white man, aged fifty-four, was taken to a hospital with fractures of the right femur and tibia suffered in an automobile accident. There was a history of preceding chronic alcoholism. The fractures were reduced under ether anesthesia and a spica cast applied. The immediate post-traumatic course was uneventful, but on the third day the patient developed signs of delirium tremens. He was given 20 gr. of sodium bromide five times a day and occasional hypodermics of morphine. He became semi-stuporous, progressively more noisy and disturbed and developed hallucinations of sight and hearing. He was disinclined to eat and his feeding was neglected. The more disturbed he became, the more bromide he was given. After ten days, his condition had become serious and I was asked to see him. The patient was semi-stuporous, noisy, incoherent, restless and in full restraint. The lids were heavy, eyes glassy, breath fetid, and tongue furred. Speech was slurring, and when the patient attempted to reach for anything (restraints had been removed during the examination) it could be seen that his arms were very ataxic. All tendon reflexes were abolished but there were no pathological reflexes. The temperature ranged between 100 and 102° F. and the pulse varied around 104. A tentative diagnosis of bromide poisoning was made. All sedatives were discontinued, a spinal drainage was done, and the patient was given saline solution intravenously and NaCl by mouth. The serology on the spinal fluid was normal. On the third day the patient was much more quiet, and from then on there was a gradual amelioration of all psychotic symptoms. On the tenth day after treatment was instituted the patient was discharged from the hospital.

Case 2.—An unmarried white woman, twenty-four years old, developed symptoms of a mild anxiety neurosis, following the unsatisfactory termination of a love affair. She was mildly depressed and disinterested, had occasional crying spells, was unable to sleep at night, and wished that she were dead. Her attending physician sent her to a hospital and prescribed 20 gr. of a bromide mixture three times a day and at bedtime. On the sixteenth day of treatment the patient became confused and hallucinated. She thought that the trees outside her window were waving at her and that voices were coming from the light socket on the wall. She was restless, resistive and could be heard occasionally to call out loudly for various members of her family who she imagined were in an adjoining room. I was asked to see her on the twenty-third day after she entered the hospital and found her in an active delirium. She was somnolent and restless, it was impossible to secure her attention, most of her answers were irrelevant and she was completely dis-

oriented for time and place. She spoke frequently of small aeroplanes she saw flying around her room. The neurological examination showed the pupils to be small but active to light. The patient was unable to accommodate. There was a fine bilateral nystagmus and the speech was slurring. All tendon reflexes in the arms and legs were brisk but no pathological reflexes were elicited. Coördination could not be tested. A diagnosis was made of bromide intoxication superimposed upon an anxiety neurosis and eliminative and supportive treatment was instituted. All sedatives were discontinued, NaCl was given parenterally and by mouth, and the patient was fed by nasal tube. I did not see her again but her physician reported that three weeks after treatment had begun all signs of delirium had disappeared and the clinical picture had returned to the original one of an anxiety neurosis. From then on, this patient's illness ran a course typical of this disease and she eventually made a complete adjustment.

Case 3.—A thirty-five-year-old white man was taken to a hospital in a distant town, suffering from chronic alcoholism. He had been drinking on an average of a quart of cheap whiskey every day for three months. For two weeks prior to his admission to the hospital he had been mildly confused and in the habit of taking long walks alone in the country. One day he was picked up in a neighboring town in a dazed condition, not knowing how he got there. He was taken to the hospital and placed on bromides and chloral by mouth. On the eleventh day his mental condition had become much worse and I was asked to see him. I found a powerfully built man lying in bed secured by restraints on all four extremities. He was yelling, cursing and shouting. Distractability was extreme and the patient was completely disoriented. Any examination was practically impossible but the patient's chart showed that his temperature was varying between 100° and 102° F. rectally and the pulse around 120-140. Very little food had been taken. The chart also showed that during the eleven days the patient had been in the hospital he had received 2,200 gr. (14.6 gm.) of bromide, 1,100 gr. (7.3 gm.) of chloral, 45 gr. (3.0 gm.) of phenobarbital and several hypodermics of hyocin. Immediate discontinuance of all sedatives was advised, and NaCl was given intravenously and by nasal tube. Restlessness was controlled by warm, moist body packs, and on two occasions it was necessary to administer ½ ounce (7.5 c.c.) of paraldehyde in an oil retention enema to control the restlessness. I did not see the patient again but the attending physician reported that he made gradual but steady improvement, and in three weeks was discharged from the hospital in good condition.

It must be admitted that in none of the above cases was the existence of bromide intoxication proven by laboratory methods. However, the fact that the patients were steadily growing worse under bromide medication, developed an acute delirious psychosis, and improved promptly and consistently upon withdrawal of the drug and replacement by chlorides, is significant. In the following three cases definite laboratory evidence of elevated serum bromide was obtained at the height of the psychoses. Blood bromide determinations were made by Miss M. Seltz, laboratory technician at Mounds Park Sanitarium in St. Paul. The LaMotte comparator was used. As a colorimetric method, this is fairly accurate.

Case 4.—A white man, sixty-nine years old, was admitted to a hospital on February 25, 1937, complaining of intense vertigo associated with attacks of nausea and vomiting. From the description of the symptoms, I believe that he was suffering from a labyrinthitis. The physical, neurological and psychiatric examinations were negative throughout. Because of a mild hypertension and some signs of renal insufficiency, the patient was placed on a salt-free, meatless diet, and was given 15 gr. (1.0 gm.) sodium bromide six times a day. To offset this amount of bromide, he should have taken 360 gr. (20 gm.) NaCl every twenty-four hours but he was on a salt-free diet. After a few days the patient's vertigo and vomiting ceased and he was in good spirits and progressing satisfactorily. By March 10 he was up and walking around but still receiving bromide and no salt. On March 12, he began to have thickness of speech and anorexia (nurse's notes). He developed fever and pain in the chest and x-ray films showed a bilateral basal bronchopneumonia. A medical consultant was called by the attending physician and his notes state that the patient was too confused to permit a satisfactory examination. Mental confusion and restlessness continued and the patient was given frequent hypodermics of dilaudid. The febrile condition lasted for about ten days and during that time the patient did not take anything by mouth, so received no bromides. After his fever subsided his mental condition persisted and I was asked to see him on March 27, 1937. He had not received any bromide for fifteen days, but he was confused, restless, disoriented and hallucinated. He expressed delusions of infidelity and many of his formulations had a marked persecutory trend. The speech was slurring and the eyes staring but the skin was clear. The patient's mental condition was such that commitment to a state hospital had been advised. In reviewing his chart, I found that the patient had received 1,350 gr. (90 gm.) of sodium bromide in fifteen days, but no NaCl. Fifteen days after the last dose of bromide, his serum bromide still was 160 mg. per 100 c.c. This is in the toxic zone. NaCl by mouth and parenterally was advised and paraldehyde was used to control the restlessness. On the fourth day the patient was eating well, but was still confused and disoriented. On April 8, eleven days after treatment was started, the patient was walking out of doors, and on April 11, two weeks after institution of NaCl and supportive treatment, the patient was discharged from the hospital perfectly clear and rational mentally.

In this case it might be argued that the patient was suffering from a toxic delirium secondary to his pneumonia. I am inclined to doubt this, first, because the pneumonia was not a very toxic one, and the delirium was present to some degree before the temperature rose. Furthermore, the delirium continued for some time after the pneumonia had subsided, it was accountable for by an excess of serum bromide and it subsided when treatment for bromide delirium was instituted.

Case 5.—A white man, aged forty-two, with a history of occasional alcoholic excesses, was greatly upset by the death of his wife on March 1, 1937. He began to drink excessively and on March 10 consulted his family physician, who made a diagnosis of chronic alcoholism and melancholia. The patient was advised to stop drinking, which he did, and was placed on 20 gr. NaBr four times a day. On March 24 his gait became ataxic and his speech slurring. By March 28, his arms also were ataxic and it was impossible for him to feed himself. He was mildly confused and, because of the mental state and the ataxia, syphilis of the central nervous system was suspected and the patient was sent to the hospital for study. I saw him on March 28 and found him to be well oriented, but very sluggish mentally. There was profound emotional depression with a strong tendency to cry at the slightest provocation. The speech was slurring, there was a fine bilateral nystagmus, and all tendon jerks were reduced. There was marked bilateral ataxia of the arms and legs. The blood Wassermann was negative and the cerebrospinal fluid gave normal serology and cytology. The blood serum bromide was 310 mg. per 100 c.c. Under NaCl treatment, moist body packs, massage, and withholding all sedatives, the patient improved gradually and was discharged from the hospital on April 20 completely recovered from his ataxia, slurring speech, and mental sluggishness, but still emotionally depressed to a modest degree.

This was a case of simple bromide intoxication and had not yet gone on to the stage of delirium or psychosis. The clinical picture was very similar to that of tabo-paresis.

Case 6.—M.P., a white woman, aged fifty-two, was first seen by me in consultation on March 3, 1937. She was suffering from a moderately severe anxiety neurosis with numerous gastro-intestinal preoccupations. The neurological and physical examinations gave normal findings. A plan of treatment was outlined and the patient made satisfactory progress, being discharged from the hospital by her physician on April 9, 1937. On April 23, I was asked to see her again because she had returned to another hospital in a state of acute delirium. She had gone home taking 20 gr. of bromide mixture four times a day. We learned from relatives that she had had a return of her gastro-intestinal symptoms and since going home had eaten very little. When I saw her the second time she was confused, totally disoriented, and noisy enough to disturb other patients in the hospital. She was extremely restless, her speech was slurring, the lids were heavy, and the eyes had a dull staring expression. The tongue was dry and furred and the breath was foul. The neurological examination, as far as could be carried out, was normal. Blood serum bromide was 275 mg. per 100 c.c. The usual treatment was instituted and at the time of this report, May 2, 1937, the patient is more clear mentally, quiet, but still restless.

Comment

It will be noted in the above cases that the patients were taking moderately heavy doses of bromide over relatively short periods of time. However, none of them was eating a sufficient amount of food and, consequently, not getting enough chloride. This probably accounts for the early appearance of toxic symptoms in these cases. The fact that toxic symptoms were present for only a short time before treatment was instituted accounts for their rapid disappearance after treatment was begun.

The question naturally arises — why, with thousands upon thousands of people receiving bromide medication, do we not encounter more cases of intoxication? That is a difficult question to answer but I believe that it can be explained, in part, on the basis of individual susceptibility. Some people have a lower "delirium threshold" than others. We all know individuals who become delirious with relatively slight rises in temperature regardless of the cause. On the fever up to 105° to 106° F. without showing any signs of delirium. Then there is the question of individual susceptibility. Some people get sick on as little as 15 gr. (1.0 gm.) of potassium iodide a day, and then there are others who can take 200-300 gr. daily without the slightest difficulty. The individual variations in susceptibility to alcohol are too well known to merit discussion here. Decreased powers of elimination due to arteriosclerosis and renal insufficiency also favor intoxication in some cases, and in others the presence of another toxic factor such as alcohol or toxemia due to infection tends to aggravate the clinical picture. I feel, therefore, that one or more of several factors are necessary to produce bromide intoxication. These are: the administration of moderately large doses of bromide, decreased chloride intake, low delirium threshold, heightened individual susceptibility, and impaired elimination or the presence of another toxic factor such as alcohol or infection.

Summary and Conclusions

1. Due to the increase in functional nervous diseases more bromides are being prescribed than formerly.

2. In susceptible individuals bromides may produce mental symptoms even where given in usual therapeutic doses.

3. The effects of bromide are largely confined to the central nervous system. Bromide tends to displace chloride in the blood stream.

4. With the administration of bromide an adequate chloride intake is necessary to prevent bromide intoxication.

5. The toxic effects of bromide may be described as simple bromide intoxication or depression, and bromide delirium or psychosis.

6. Blood serum bromide in excess of 150 mg. per 100 c.c. is in the "toxic zone."

7. The duration of symptoms of brominism following discontinuance of the drug is two to six weeks.

8. Treatment of bromide intoxication consists of discontinuing the drug and all other sedatives, the administration of NaCl orally and parenterally, hydrotherapy, and, in urgent cases, quick acting and rapidly eliminated sedatives. Spinal drainage sometimes helps, and gastric aspiration has been recommended to help eliminate the bromide.

9. Three probable cases and three proven cases are briefly reported.

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ACUTE PULMONARY EDEMA OCCURRING DURING PREGNANCY OR LABOR*

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ACUTE pulmonary edema as a terminal complication of pregnancy or labor is a most serious one and a rather frequent complication of eclampsia, occurring in almost one-third of the eclamptic cases. Its incidence in eclampsia without convulsions is even higher.

Acute pulmonary edema occurs late in the course of pre-eclamptic and many fatal cases of eclampsia, and during labor in cases with cardiac decompensation without audible valve lesions. It has been suggested that it may develop in the fatal cases of eclampsia as an end-result of the failure of the pulmonary circulation. On the other hand, the fact that not all cases of pulmonary edema end fatally seems to exclude this explanation.

Slemons[4] found acute pulmonary edema in six of seven cases of eclampsia without convulsions which came to autopsy.

King, et al,[3] report a series of thirty cases of ante- and intrapartum eclampsia with four cases of edema of the lungs with three recoveries. The fatal case was a colored woman, who had marked pulmonary edema on admission. The edema was relieved by appropriate treatment. She was delivered by a very easy low forceps operation seventeen hours after admission. Through a misunderstanding, ether was used as the anesthetic with a recurrence of the edema, broncho-pneumonia and death. The edema has been reported in all stages of pregnancy from the second month[1] through to term, during delivery[6] and in the puerperium. However, its greatest incidence is after the fifth month of pregnancy. It is more frequent in multiparas than in primiparas[5] and

*From the Obstetrical Department, St. Cloud Hospital. Read before the joint meeting of the Stearns-Benton and Upper Mississippi medical societies at Little Falls, Minnesota, October 21, 1937.

rarely seen in non-pregnant subjects. While it may complicate many types of cardiac lesions, in most cases it follows mitral disease, and many patients with mitral lesions have no disturbance during pregnancy or labor. In some reported cases, however, there has been no history of cardiac symptoms before pregnancy.

Pathogenesis

Bustos Moron[1] summarizes the old theories of pathogenetic mechanism of this conditions as follows: (1) a mechanical disturbance, pulmonary hypertension and acute weakness of the right ventricle; (2) a reflex disturbance originating in aortitis and peri-aortitis; and (3) a toxic element which is always present. These are all elements which collaborate in the production of the syndrome. On the other hand, today the mechanism of the attack is considered due to a severe left ventricular insufficiency. Bustos Moron believes, along with Waldorp, that within a few seconds after the occurrence of the left ventricular defect, a sudden pulmonary flux is produced, the whole parenchyma becomes congested from vertex to base, and the serous transudate causes edema of the lung. According to my observation, the edema comes on in a few minutes after the initial cough. There is a marked bronchorrhea, fine sub-crepitant and bubbling râles with some impaired resonance. This confirms the diagnosis made on the basis of the facies and the repeated cough, with an abundant or scanty yield. The pulse is lost, there is an increase in frequency and sudden hypertension, the dyspnea increases to orthopnea, consciousness may be lost, and death may follow unless proper therapeutic measures are instituted.

Symptoms

The onset is sudden, the cough persistent, and there is a sense of thoracic constriction soon followed by increasing dyspnea and precordial pain. There is marked cyanosis and spumous, salmon-colored expectoration containing blood. Fine râles may be heard at the lung bases and later throughout the lungs of the bubbling type.[2] The increased respiratory sounds obscure the heart tones, but a systolic murmur or a diastolic rumble may be heard at the apex. The pulse is rapid and thready or may be hard and tense, especially in the eclamptic type. The blood pressure is variable, the differential pressure tending to diminish. The temperature is normal or even subnormal. Albuminuria will be present at the end of the attack even in the absence of a pre-existing nephritis.

Types

Szer[5] distinguishes three clinical types of acute pulmonary edema in pregnancy. In the acute type, suffocation is intense and the patient may die within a few minutes with a pink froth on her lips. In the bronchoplegic type, expectoration is absent but the patient has severe dyspnea with thoracic pain. Signs of cardiac weakness appear rapidly. The patient does not cough and fluid accumulates in the bronchi and lungs. Cyanosis is marked, the blood pressure decreases and death may follow quickly in spite of treatment. The subacute forms are most frequently encountered. These are characterized by a nocturnal pseudo-asthma, described as a cardiac asthma by some authors, which awakens the patient. There is dyspnea with pink, foamy expectoration. Examination will reveal localized areas of edema. These attacks may be repeated frequently at night until the acute form appears.

Diagnosis

The typical forms are readily diagnosed. Diagnosis is based on the sudden onset of cough, the intensity of the edema, the characteristic edema and the auscultatory signs.

The condition may be confused with asthma, but in the latter there is a history of a previous attack and the dyspnea is of the expiratory type. Expectoration appears only at the end of the attack. The sputum is thick and contains eosinophiles. Pulmonary embolism and suffocating pneumothorax must be excluded in the diagnosis of the bronchoplegic type and this is affected by the pain in the side and the absence of expectoration.[5]

Pathology

In an autopsy reported by Teel, et al,[6] the gross and microscopic findings were interpreted as indicating that the patient suffered from a true acute toxemia of pregnancy. The pathologic lesions in the liver, kidney and brain were similar to those found in fatal cases of eclampsia, except that they were less marked. The findings in the heart and lungs were interpreted as indicative of "acute left ventricular failure, resulting in marked pulmonary congestion and edema

with slight emphysema." The heart weight (335 grams) and the subendocardial fibrosis of the myocardium and the mitral leaflets were insufficient to justify a diagnosis of chronic heart disease. No organism could be demonstrated in the myocardium or grown from a spleen culture.

Prognosis

The immediate prognosis is based primarily upon the amount of expectoration and the condition of the blood pressure but also depends largely on the promptness with which treatment is instituted. A large amount of expectoration is a favorable prognostic sign. Gallop rhythm, alternating pulse and a decrease in the differential blood pressure indicate insufficiency of the left ventricle and, consequently, offer a poor prognosis. In the presence of persistent râles at the bases, high diastolic pressure and arrhythmia, a guarded prognosis is indicated.

If the patient survives delivery and the early puerperium, the ultimate outlook as regards chronic cardiovascular and renal disease is good. Teel, et al, reported six cases, with two deaths. Three of the surviving patients were alive and well without cardiac, vascular or renal sequelæ, eight, six and two years after the attacks. King, et al, report four cases with one immediate maternal death.

Treatment

The basis of the treatment of acute pulmonary edema is heroic doses of morphine with atropine and heart stimulants, with venesection to reduce the venous hypertension and relieve the pulmonary circulation. Immediate relief follows the removal of 500 to 700 c.c. of blood. Peripheral venostasis has been recommended as an alternate to venesection.

Cardiac weakness indicates a cardiac tonic, as ouabain, which acts rapidly when given intravenously. Assuming that the attacks are similar in mechanism to classical cardiac asthma, Teel, et al, feel that digitalization is indicated. These same authors also believe that the prophylactic use of digitalis in all eclamptics and patients with severe non-convulsive toxemia in and above the fourth decade of life should be considered. The results obtained by digitalization in the severe decompensated hearts at times are not what one would expect even with larger doses than that usually given.

From the obstetrical viewpoint, Szer believes that, while there is no fixed rule, early interruption of the pregnancy is indicated in those cases in which signs of grave cardiac insufficiency are present at the onset of pregnancy. Cesarean section should be considered when serious attacks occur repeatedly in spite of medical treatment. From a limited experience, Teel, et al, concluded that significant clinical improvement was not to be expected until the uterus was emptied. The optimum time for interference appears to be between twelve and forty-eight hours after the attack. This delay gives sufficient time for digitalization before the interruption of the pregnancy is undertaken.

Delay, as suggested by Teel, et al, may be tried in patients with a favorable cardiac condition and a mild lung involvement. The edema, however, may become suddenly severe and terminate fatally. In the more severe cases, the treatment must be instituted promptly and the uterus emptied as soon as possible. The bleeding after delivery is profuse and appropriate treatment must be instituted to control same.

When acute edema of the lungs occurs towards the end of pregnancy, it is often followed by labor. If necessary, dilatation may be completed manually and delivery effected by means of forceps or version.

Case Reports

Case 1.—Mrs. J. T. L., gravida 1, aged twenty-seven, had had the diseases of childhood, smallpox, tonsillitis, influenza, tonsillectomy, dyspepsia with urticaria of one week duration in 1932, Schoenlein's disease with a kidney complication and mitral insufficiency following an upper respiratory infection in 1923. Her last menstrual period was December 25, 1926, and the date of expectancy was calculated for October 1, 1927. The family history was negative. Edema of the lower extremities appeared when she was six months pregnant. Because of financial difficulty, she continued with her professional work as a nurse, which necessitated being on her feet a great deal. She was advised to discontinue working but refused. October 1, 1937, she was admitted to the hospital with a generalized edema and a mild respiratory embarrassment, temperature 98.2, pulse 80, respirations 28, blood pressure 160/120. Urinalysis of a catheterized specimen showed sp. gr. 1.014, acid, albumin 2 mm. ring, acetone, diacetic acid, R. and W. B. C. Other physical findings were negative including examination of the heart.

She was given castor oil and a hot pack to stimulate elimination; spontaneous rupture of the membranes occurred four hours later. Her progress was slow and the patient was in labor forty hours. Morphine and

scopolamine with ether in oil per rectum and magnesium sulphate intramuscularly were used during the first stage of labor. The head remained high in the right transverse position; the cervix was about two-thirds dilated. Suddenly the patient developed a cough with râles in both lungs, temperature 98.3, pulse 114, respirations 48. Appropriate medication was given and the patient prepared for delivery. Labor was terminated by manual dilatation of the vagina and cervix, with a difficult high forceps operation. The baby was in fair condition. A severe postpartum hemorrhage occurred after the expulsion of the placenta. The uterus was packed and 1 c.c. each of pituitrin and ergot were given by hypo. The estimated blood lost was 750 c.c., pulse 118, respirations 50. The patient's condition was poor. There was considerable bleeding the first postpartum day partially controlled with ergot. The labored breathing continued for twenty-four hours. The lungs were free of râles by the fifth postpartum day. The patient was able to leave the hospital on the thirteenth postpartum day. A subsequent pregnancy fifteen months later was normal throughout.

Case 2.—Mrs. J. L., gravida 4, aged forty, had her last menstruation August 4, 1928. She had had rheumatism in 1923, spontaneous full-term delivery in 1927, and rheumatism in 1928 when two months pregnant. Urinalysis at this time showed albumin, hyaline and granular casts, R. and W. B. C. No blood pressure readings were recorded at this time by the attending physician. There was no prenatal care.

The patient was first examined by me April 11, 1929. She was in labor and she was coughing, breathing laboriously, cyanosed, and sitting up and stooped forward. Crepitant râles were heard in both lungs, and a systolic heart murmur heard at the apex and transmitted to the axilla. Morphine 0.01 (gr. 1/6) with atropine 0.0004 (gr. 1/150) was given with relief in one-half hour. The patient was prepared for delivery, anesthetized, catheterized, the cervix dilated manually, and forceps applied to the head in L. O. A. position. Delivery was easily accomplished. The baby was thin but in good condition. The lungs were clear after forty-eight hours. The patient made an uneventful recovery following her delivery. The heart and kidney condition remained unchanged. The patient was up and about the tenth postpartum day.

On May 9, 1929, while sitting at the table after supper, she was suddenly seized with a severe attack of coughing with labored breathing, cyanosis, expectoration of a serous, frothy, blood-tinged fluid (about 500 c.c.). Bubbling râles were heard in both lungs, the heart was rapid and a systolic murmur was heard over the aortic area which was transmitted into the neck, and a systolic murmur over the heart apex transmitted into the axilla. Morphine 0.01 (gr. 1/6), atropine 0.0004 (gr. 1/150), were given by hypodermic and the atropine had to be repeated in a half hour before the patient got any relief from the edema of the lungs. The heart was digitalized, and the morphine and atropine had to be given daily for a week before the lungs remained clear. The swelling gradually became generalized with accumulation of fluid in the peritoneal cavity. The urinary output diminished until there was complete suppression. The patient expired February 19, 1930, from cardio-renal dropsy. Necropsy was not obtainable.

Case 3.—Mrs. J. W., gravida 2, aged thirty-three.

The patient was seen in consultation and referred by Dr. A. Mahowald. At the age of thirteen, she had had pain during and after urination for three or four days. She had had many of these attacks up to the time of her marriage seven years ago. She had had measles at the age of twenty-one, and edema of the lower extremities during the last month of her first pregnancy. She went to term and was delivered by an easy forceps operation. She had her last menstrual period March 25, 1930. The date of expectancy was January 2, 1931.

About one month prior to the development of the complication, she was examined by her physician for swelling of the lower extremities. On the afternoon of January 1, 1931, she developed a mild cough with free expectoration of fluid which gradually increased in amount. At about eleven P. M., the patient was awakened, smothering, with fluid escaping from her nose and mouth, coughing severely and breathing with difficulty. The family physician was called and he immediately took her to the hospital. She did not remember anything other than riding fast in a car. She was admitted to the hospital at 12:55 A. M.. Her physician ordered a hot pack and an enema, and magnesium sulphate was given by mouth, but was not retained. At 3:10 A. M., she was given 0.03 (gr. ½) codeine with some relief for one hour. At 4:30 A. M., he took her to the delivery room and packed the cervix with sterile gauze to induce labor, but was unsuccessful. The patient was seen by me at 7:30 A. M. She was sitting up in bed supported by a back rest. Serous blood-tinged fluid was escaping from the mouth and nose; she was coughing and vomiting simultaneously and struggling for air. The patient was cyanotic, cold and clammy, and there were bubbling râles in both lungs. The heart tones were regular, rapid, and difficult to detect; the pulse 120, respirations 42. Urinalysis: Albumin 2 mm. ring; hyaline and granular casts, R. and W. B. C. Morphine 0.01 (gr. 1/6), atropine 0.0004 (gr. 1/150) was given by hypo, and the atropine was repeated in ten minutes. The lung condition cleared sufficiently to allow the patient to be moved to the delivery room and prepared for delivery. The patient was placed in a semi-Fowler position; oxygen was given for the cyanosis; she was anesthetized with N_2O. After catheterization, the cervix was dilated manually, forceps were applied to head in R. O. P. position and the head was so delivered. The baby was in good condition. The patient was given ergot 1 c.c. per hypo to stimulate uterine contraction. Oxygen was given for the cyanosis. The placenta was expressed. Some 500 c.c. of blood was lost after the placenta was expelled. The tendency to bleed was marked and ergot had to be repeated in an hour. Two hours after delivery atropine was given for recurrence of the edema. The patient coughed a great

deal the day of delivery, with free expectoration of bloody mucus. The blood pressure after delivery was 140/100, the temperature 101, pulse 100, and respirations 40. The progress was uneventful after the second day and the patient was discharged on the eleventh postpartum day. The blood pressure on the day of discharge was 110/70.

Her third pregnancy was without complication. She had her last period on December 25, 1931, and delivered spontaneously on August 28, 1932, one month earlier than the date of expectancy. A trace of albumin with moderate swelling occurred at the end of the seventh month with a blood pressure of 150/110. The blood pressure returned to normal and the urine was free of albumin following rest, diet, and the limitation of the fluid intake. Her weight the day of her first visit, February 28, 1932, was 182 pounds and the blood pressure 110/70. The day of her delivery her weight was 189.5 pounds and B. P. 130/88.

Her fourth pregnancy in 1936 was normal throughout.

Case 4.—Mrs. H. L., gravida 6, aged thirty-six, had had five full-term pregnancies, of which four were normal. During her fifth pregnancy, on the day of delivery, she developed a severe headache, vomiting, and numbness in the lower extremities. Her blood pressure was 160/100. The urinalysis of a catheterized specimen showed albumin 2 mm. ring; sp. gr. 1.020, hyaline and granular casts, R. and W. B. C.

She had had her last period September 16, 1930. The date of expectancy was June 23, 1931. The heart, kidney, and blood pressure were normal throughout the gestation. During the week prior to entering the hospital, she had respiratory difficulty which would occur from one to two hours after lying down at night. She was examined about midnight, June 7, 1931, in her home. The patient was delirious and did not recognize her family. She was sitting up and struggling for air. There were many râles in both lungs; no heart murmurs could be heard. Her temperature was 98, pulse 128, and respirations 30. She was taken to the hospital and admitted at 12:10 A. M. A non-catheterized specimen of urine showed a trace of albumin. Blood examination: R. B. C. 4,250,000; W. B. C. 9,400; Hbg. 76%. Codeine sulph. 0.03 (gr. ½) was given for the relief of the edema of lungs and restlessness. The patient was relieved by the medication and was in fair condition in the morning. At ten o'clock A. M., the membranes were ruptured artificially. Castor oil and quinine were given. Contractions started three hours later, and she was in labor one hour and ten minutes before the baby was born. Fifteen minutes prior to delivery she suddenly developed cyanosis, a severe cough, labored breathing, and râles in both lungs. Her pulse was 160, and respirations 32. The patient was placed in a semi-Fowler position. Atropine 0.0006 (gr. 1/100) with whiskey 15.0 (oz. ½), pituitrin 0.3 (Mv.) were given to hasten delivery. The baby was delivered spontaneously in good condition. The placenta was expelled by the Credé method. The patient's condition was fair when she was taken to her room. She was placed in Fowler's position. Digitalis was given for the heart, oxygen for the labored breathing and cyanosis, morphine sulphate 0.01 (gr. 1/6) for restlessness and cough and ergot 1 c.c. to check the severe bleeding. After the fifth day the lungs were clear. The patient left the hospital on the eleventh postpartum day. The pulse was 96 and respirations 28. The patient's progress was slow and stormy. The heart was digitalized but never responded well to treatment. She died suddenly August 17, 1933, when she left her bed for elimination purposes.

Case 5.—Mrs. L. R. B., gravida 1, aged twenty-four, had had her last menstruation May 31, 1936. The estimated date of delivery was March 6, 1937; the date of delivery February 27, 1937. She was in labor sixteen hours.

This patient had had scarlet fever, measles, pertussis, diphtheria, chorea and rheumatism. She had had two attacks of rheumatism at the age of nine and sixteen years, her second attack lasting sixteen weeks. An appendectomy had been performed at the age of nineteen.

Physical examination on October 14, 1936, showed a well-nourished female sixty-seven inches tall, weighing 199.5 pounds. Her blood pressure was 170/70. All other physical findings were normal except that the heart was enlarged and a systolic murmur could be heard over all of the valve areas. She was about five months pregnant and her pelvis was large. The kidneys remained normal throughout the gestation and she gained 17.5 pounds from the time she presented herself for observation to the time of delivery February 27, 1937. The blood pressure dropped to 150/70 and gradually rose to 190/60 the day of delivery. The first pains came on about 3 A. M. and she entered the hospital at 7:15 A. M. Castor oil was given to stimulate contractions. On rectal examination, the head was mid-pelvic, the cervix thinned out and dilated about 2 cm. The fetal heart tones were heard to the right and below the umbilicus, 150 per minute.

The patient's heart condition was satisfactory and she was given her first dose of morphine and scopolamine at 2:05 P. M. At 3:15 P. M. she developed a slight cough which came at frequent intervals and fine subcrepitant râles were heard in both lungs; pulse 118, respirations 24. She was given atropine sulphate 0.0006 (gr. 1/100) with improvement. At 7 P. M., she had a recurrence of the lung symptoms and digitalin 0.0013 (gr. 1/50), strychnine 0.0016 (gr. 1/40) atropine 0.001 (gr. 1/75), were given by hypo. The atropine was repeated in twenty minutes. By this time the patient was cyanotic, the skin clammy, breathing labored, pulse 140 and there was free expectoration of blood-stained serum. She was taken to the delivery room and oxygen was given while being prepared for delivery. Under nitrous oxide anesthesia the perineum was dilated. The head in R. O. A. position was delivered by an easy forceps operation, the shoulders and body were delivered by the Christeller maneuver, the cord was clamped and cut, and the placenta expressed. One c.c. each of ergot and pituitrin were given per hypo

to control the uterus. Some 400 c.c. of blood were lost. The oxygen was started as soon as the child was delivered and kept up for fourteen hours before the cyanosis and lung edema began to subside. The heart stimulation had to be repeated at regular intervals, with morphine for restlessness and ergot to control the uterus. The labored breathing, edema and cyanosis gradually cleared and after forty-eight hours the patient was symptom-free, with the pulse returning to normal. The heart stimulation was continued and the patient was discharged the tenth postpartum day. The digitalis was kept up for two months and her final check April 15, 1937, showed the B. P. 150/60 with the heart condition improved so that the patient was able to resume her household duties.

Case 6.—Mrs. J. M. A., gravida 3, aged thirty-three, had had edema with her two previous pregnancies. Her complaint at this time was difficulty in breathing, especially when she was lying down. This condition, which she attributed to the varnishing of her home, had been of two weeks duration. When moving about she had less difficulty in breathing.

The patient was seen in the home by Dr. C. B. Lewis and taken to the hospital. The edema and cyanosis were generalized and there was morbid dyspnea, with many moist bubbling râles and expectoration of frothy, blood-stained, serous fluid. Her B. P. was 160/90, pulse 100, and respirations 30. Labor was induced and the baby was delivered with forceps. The patient's condition was poor. Cardiac stimulation and oxygen were given but she expired one and one-half hours after delivery. Bedside notes show that severe postpartum bleeding continued up to the time of death.

This case is reported with the permission of Drs. C. B. Lewis and W. L. Freeman of St. Cloud, Minnesota.

Conclusions

1. The strain of labor added to a weakened heart musculature with or without the presence of a valvular lesion is a definite factor in causing acute pulmonary edema.
2. With appropriate treatment before, during and after delivery, maternal mortality from pulmonary edema should be reduced to a minimum.
3. Severe bleeding occurred in five of the six cases and was responsible for one maternal death.
4. With proper antenatal care in subsequent pregnancies, a recurrence of the pulmonary edema may be prevented.

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HYPERPYREXIA IN THE NEWBORN

Report of Infant With 107° F. by Rectum at Age of Fifty-three Hours

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HYPERPYREXIA of 107° (rectal) in a newborn is unusual enough to warrant detailed analysis. In the case to be reported, this temperature was observed at the age of fifty-three hours. In the newborn a temperature as high as 104° (rectal) on the third day, or thereabout, is not necessarily associated with an unfavorable prognosis. In fact, in many a newborn moderate fever is transitory and recovery occurs without special treatment. The causative factors of transitory fever have been the subject of a great deal of controversy. Infection, inanition, dehydration, and birth trauma have been considered.

Salmi summarized the literature in 1935 and reported his observations in a series of infants delivered normally, by cesarean section, and by forceps. Transient fever of 100° to 104° with onset from the second to fifth day occurred in 20 per cent of the 225 infants of normal delivery. Long labors (over twenty-five hours) were three times as frequent in the infants manifesting fever as in the fever-free group. The birth weight was above 3,500 grams in 60 per cent of the cases showing fever, whereas only 30 per cent were above this weight in the fever-free group. The incidence of transitory fever in the newborn was increased when the membranes had been ruptured early (ten hours before delivery), when meconium was present in the amniotic fluid, or when vaginal examinations had been done.

Twenty-five per cent of infants delivered by cesarean section and 50 per cent of those de-

livered by forceps showed this phenomenon of fever. Salmi calls our attention to the fact that labor had been in progress a long time in many of the cases delivered by these two methods. Thus, long, hard, complicated labors *predispose* to temperatures ranging from 100° to 104° in the newborn.

Case Report

Baby boy F., weighing eight pounds, four ounces, was born March 26, 1936, at 5:15 p. m., the first baby (first pregnancy) of a mother nineteen years of age. Both mother and father were in good health. Labor was of fifteen hours' duration and normal except for an episiotomy. Nembutal (4½ grains) and scopolamine (1/200 grain) analgesia was used during labor and nitrous-oxide gas anesthesia during delivery. The amniotic sac ruptured one hour before the birth of the child. The amniotic fluid was definitely stained with meconium.

Mucus was removed from the nose and throat by suction with a soft rubber bulb before the infant made perceptible respiratory movements, and later, in the nursery, mucus was removed again in the same manner. During the first twenty-four hours after birth the weight loss was 4½ ounces and the temperature went from normal at birth to 102.2° rectal. During the second twenty-four hours the baby received 12 ounces of complement and water by mouth and 3 ounces of 5 per cent glucose in normal saline by rectum. During this twenty-four hours the rectal temperature ranged from 100.2° to 102.2° and the weight loss was 9½ ounces. This occurred without diarrhea or vomiting being present. At the age of 53 hours the baby's rectal temperature went up to 107°.

Examination at this time showed a somewhat spastic child, very hot to touch, with respirations 90 per minute. The thighs were flexed on the abdomen. The abdomen was slightly distended but no abnormal findings were noted on palpation. There was a slight areola of redness around the umbilical cord. The fontanelle was boggy but was not bulging, and it did not move with respiration. The mucous membranes of the nose, tongue and mouth were dry. The lungs were negative to percussion and auscultation. The heart was very rapid, estimated 200 beats per minute. Dehydration was obvious, and serious respiratory disease or cord infection was suspected.

Treatment: Whole blood (10 c.c.) and normal saline (100 c.c.) were given subcutaneously. One or both of these fluids were given in like manner every three or four hours. One ounce of 5 per cent glucose in water was given by gavage every three hours. During the first twenty-four hours of this treatment, the baby received 40 c.c. of whole blood from the mother and the father, and 550 c.c. of normal saline subcutaneously and 240 c.c. of 5 per cent glucose by gavage. The whole blood and normal saline were absorbed very rapidly. The respirations and heart rate came down to normal and the temperature dropped to 101.4° rectal within twenty-four hours after the beginning of this treatment. The weight curve was interesting. In spite of the fact that 15 ounces of fluid had been forced by mouth and rectum, the baby lost 14 ounces in weight in the fifty-three hours from birth until the time the temperature went up to 107°. The baby was not disturbed during the succeeding twenty-four hours, but forty-eight hours after subcutaneous fluid was started the baby had gained 17 ounces.

Breast milk was started twenty-four hours after the beginning of the subcutaneous fluids. The milk was expressed from the mother and given by bottle every four hours. The baby nursed well when it was put to breast forty-eight hours later. Both baby and mother were discharged from the hospital on the tenth day in good condition, the baby weighing 8 pounds 7½ ounces, or 3½ ounces over birth weight.

The areola about the cord disappeared but the cord remained attached for several days after leaving the hospital. An umbilical secretion was obvious for four months, during which time a slight hernia developed. This healed by strapping with adhesive tape. Examination at six months showed a perfectly normal child.

Summary

1. A large first-born baby developed a temperature of 107° rectal at the age of fifty-three hours.

2. There was marked dehydration, possibly respiratory or cord infection.

3. Rapid and complete recovery followed treatment with whole blood and normal saline subcutaneously, and water and glucose by gavage.

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CASE REPORTS

ABDOMINAL PREGNANCY AT EIGHT MONTHS*

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THE literature on abdominal pregnancy was carefully reviewed by Cornell and Lash† (1933). In their series of 236 cases taken from the literature, their private cases, and the records of the Cook County Hospital, they reported that only 35 per cent were diagnosed correctly before operation. It is felt, therefore, that the presentation of another of these unusual cases is warranted, inasmuch as considerable diagnostic help may be obtained by use of the cystogram and the other usual x-ray studies.

In the cases reported by Cornell and Lash, the condition was found most frequently in the first and second pregnancies. They found that in the eighty-six cases in which the baby was born after six months, the infant mortality was 22 per cent, whereas in the sixty cases in which the baby was born alive in the eighth and ninth months, the infant mortality was about 35 per cent. They found a maternal mortality of 14.3 per cent (thirty-four cases) in the total of 236 cases. Cornell and Lash concluded that removal of the placenta in toto is best when the placental blood supply can be ligated and when the site of the placenta is not attached to a vital organ. Also, they advised, when necessary, to combine marsupialization with drainage.

H. O., a white female, aged twenty-eight years, was admitted to the Minneapolis General Hospital September 8, 1933. The patient had had no prenatal care. She was a gravida ii, para i. Last normal menstruation was December 16, 1932, of four days duration. January 16, 1933, she flowed one day. February 22, 1933, she passed two small blood clots, then had no bleeding at all until four days before admission to the hospital.

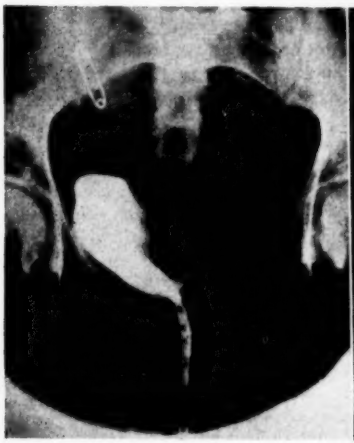

Fig. 1. Cystogram, taken after 50 c.c. of 12 per cent sodium iodide were injected in the bladder. The bladder is distorted, its mid and left portions are flattened out.

Fig. 2. Anterior posterior radiograph, showing the fetus in the left lateral portion of the abdomen.

Fig. 3. Lateral radiograph of the abdomen, showing the breech presenting.

The patient felt life June 1, 1933, and, she thought, until the day of hospital admission. She had never had any severe rupture-like pain, nor shoulder discomfort. Starting four days before admission at about 9:00 A. M., she had had moderate abdominal cramps coming about every two hours, bleeding a little less than that of a normal period, and frequency without pain or burning, to the extent of voiding about every forty-five minutes. Her bowels had moved that day, the stool being a little constipated, but not more so than usual.

Examination showed blood pressure 116/68, temperature 98.8°, pulse 78 and respiration 20. There was secretion from the nipples. A symmetrical pelvic mass was present on the right side extending within 3 cm. of the navel. The abdomen was soft, showed no peritoneal irritation, no fluid, no shifting dullness. On the left side of the abdomen there was a mass extending up to the costal arch. The upper pole felt

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†Cornell, Edward L., and Lash, A. F.: Surg., Gynec. and Obst., 57:98, 1933.

like a fetal head, and could be palpated distinctly. The lower pole lay just above the pubic bone and could not be palpated distinctly. No fetal movements were felt nor heart beats heard. Pelvic examination showed a little sanguinous discharge from the vulva, floor was competent, and there was no evidence of

Dr. J. H. Simons and the writer. A lower midline suprapubic incision was made. Examination of the abdominal cavity showed the omentum to be attached to the mass in the right lower abdomen. The mass was the encapsulated placenta. The omental blood supply to the placenta was ligated in dissecting out

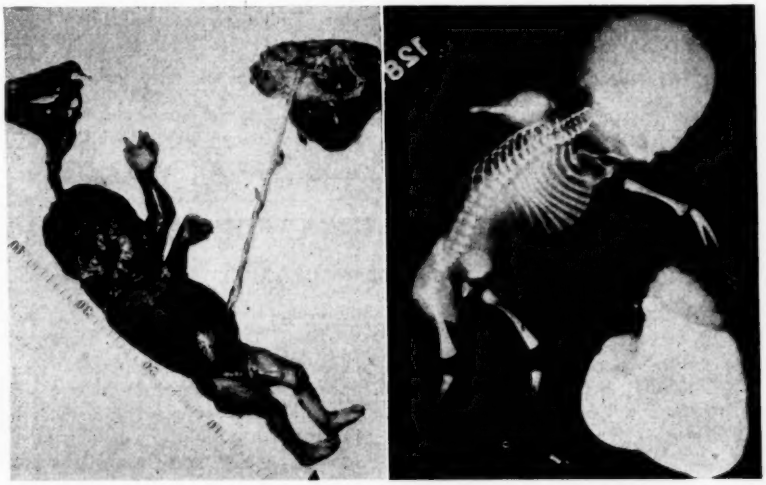

Fig. 4 *(left)*. Photograph of baby and placenta, showing the baby's length and maceration.
Fig. 5 *(right)*. Radiograph of baby, showing no evidence of syphilis in the long bones.

infection. Vaginal walls and cervix were only slightly blue, and the cervix was a little soft. There was a little bleeding from the cervix, about like that of a normal period. A symmetrical uterine-like mass, about the size of a three months pregnancy, occupied the right pelvic fossa. The breech, which lay to the left of the inlet, displaced this uterine-like mass to the right. Nothing abnormal was palpable in the cul-de-sac or either adnexa, but the left adnexa was a little tender.

A cystogram was taken after injection of 50 c.c. of 12 per cent sodium iodide in the bladder. The picture showed the bladder to be distorted, its mid and left portions being flattened out, apparently by the breech.

Anterior posterior and lateral radiographs were made, which demonstrated a single fetus lying anterior posterior and slightly transverse in the left lateral portion of the abdomen with the head to the left of the midline anteriorly opposite the first, second, and third lumbar vertebræ, and with the back posterior. There was a marked over-riding of the cranial bones, apparent collapse of the calvarium, and accentuation of the curves, indicating that the fetus was dead. The position of the fetus was one of extreme abnormality. Its density coincided with the continuation of the curves of the uterine mass, and its appearance definitely indicated that it was outside of the uterus.

The laboratory analysis showed hemoglobin 73 per cent; white blood count 10,500; polymorphonuclears 73 per cent; lymphocytes 25 per cent; and monocytes 2 per cent. Urine was entirely normal.

A preoperative diagnosis was made of:

1. Probable left extrauterine pregnancy with myomatous uterus or

2. Incarcerated dead fetus in a myomatous uterus.

Operative Record.—The patient was operated on by the placenta from its capsule. Further examination showed the uterus to be the size of a three months pregnancy lying in the true pelvis. The placenta was a trilobed mass adherent to the posterior abdominal wall, intestinal coils, and omentum. The placenta rested just above the uterus, but distinctly separate from it, and a little to the right of the midline. The lower portion extended over the brim of the pelvis posteriorly. Examination of the adnexa showed both ovaries to be normal. Both tubes appeared slightly thickened, but otherwise they also were normal. The appendix was not observed. Examination showed a dead female fetus in the left abdomen. The fetus had probably been dead only a short time, as there was very little maceration. The baby lay anterior posterior with head anterior, back posterior, and breech presenting. The amniotic sac had not ruptured. There were many adhesions between the amniotic membranes and viscera. The sac was firmly attached to the baby's head. Almost the entire area of the amniotic sac was adherent to intestines and abdominal wall, but these adhesions were easily released. The omentum, containing large blood vessels, was attached to the anterior surface of the placenta, to the ascending colon, descending colon, and small intestine. The cord extended from the opening on the left side of the placental sac over intestinal coils to the fetus. The placenta obtained most of its blood supply through the large omental vessels, and lay in a capsule of two portions, a rather thin amnion and an outer thick membrane.

The fetus and membranes surrounding it were liberated and the baby extracted with the cord. There was no line of cleavage to the outer thick placental sac, so this was opened and the placenta peeled out. There was considerable bleeding which was easily controlled by temporary packing. This thick sac remaining after the placenta was removed was cut down to its mesenteric attachments, leaving enough to approximate the edges together with a running suture. A Penrose drain

was inserted to take care of any oozing that might occur. After ascertaining that there was no free bleeding, the abdomen was closed.

During the operation the patient's blood pressure went down to 70/30. 1,000 c.c. of 10 per cent glucose solution and 600 c.c. of citrated blood were given in the right cubital vein. The pressure went up to 110/70, and the patient was returned to the ward in good condition.

The fetus measured 41 cm. crown heel, 28 cm. crown rump, and weighed 1,355 grams. The cord was 35 cm. long. The placenta weighed 735 grams, was the size of a grapefruit, and was composed of three equally large lobes.

Microscopic section of the placenta showed chorionic villi and trophoblasts with blood clots. Rather large areas were hyalinized and necrotic. The surface at one point was infiltrated with lymphocytes and eosinophiles. The microscopic diagnosis was placental tissue with infarcts.

The maternal blood came back Wassermann-positive, Kahn 4 plus. This was repeated with similar findings. The patient gave no history of syphilis, and had had no treatment. The baby had been dead long enough so that no blood was obtainable for the Wassermann test. Radiograph of the baby's long bones showed no evidence of syphilis.

The patient made an uneventful recovery, running a low grade postoperative temperature for five days, the highest reading being 100.4° on the day following operation. The Penrose drain was removed on the third day, and the patient was discharged on the fifteenth day.

ACUTE LYMPHATIC LEUKEMIA WITH REMISSION

H. G. BOSLAND, M.D.

Verndale, Minnesota

J. P., a white female, aged three, was first seen December 25, 1936, for what seemed to be an attack of upper respiratory infection with influenzal manifestations. She was obviously quite ill with high fever, rapid pulse and chills. Onset had occurred two days previously with the usual symptoms of a "head and chest" cold. The other members of the family were just recovering from the "flu."

Physical findings were acute tonsillitis and pharyngitis, a moderate bilateral cervical adenitis entirely commensurate with the throat pathology, a few rather coarse râles in either lung, temperature 103 by axilla, and a general appearance of pallor combined with what seemed to be a state of excellent nutrition but which also gave the appearance of being abnormal as a kind of generalized tissue swelling of a resilient type. The mother stated that all of her children and particularly this one had been large, heavy, and well-nourished as infants. The urine was normal.

The father, mother, one sister and two brothers are all living and well. There had been no deaths in the family. The mother had gone to full term with all of her pregnancies and had had normal deliveries. There had been no miscarriages nor stillborn infants.

Treatment was symptomatic for fever and restlessness, ice packs to cervical glands, mustard plasters to chest, swabbing of throat, and hot drinks. The following day the temperature was down to normal, the patient had rested well and appeared much improved clinically. No thought of leukemia at this time entered my mind, my diagnosis being acute tonsillitis and an upper respiratory infection.

The patient was not seen again until January 30, thirty-five days later, at which time the father called me because the child's "cold" had become worse again. The interval history revealed that she had not seemed to recover her usual strength and vigor and was often irritable and peevish. She had continued to have a watery rhinorrhea and an occasional slight fever but otherwise seemed well in that her appetite was fairly good and she played. Two days previous, she had become definitely worse. After a short examination, it was decided to take her to the hospital. Her temperature was 104.2 (r). An acute pharyngo-tonsillitis was present with a red angry looking patch on the right anterior pillar covered by a whitish exudate. The cervical nodes had increased in size, especially in the sub-mandibular region on the left, where the mass was very firm, somewhat tender, fixed, and seemed to represent a coalescence of several nodes. The ears were normal. The chest was normal on physical examination and x-ray. The urine was normal. Examination of the blood revealed a white count of 1,300, red count 2,450,000, Hgb. 30 per cent. In the differential, no pmns. could be identified with certainty. The predominating white cells were young lymphocytes. A blood smear was submitted to the pathology department of the University of Minnesota, the report returning with a diagnosis of acute lymphatic leukemia.

Symptomatic therapy was instituted: liquid diet, ice collar, sedatives. Liver extract was given parenterally in the hope that it might combat the neutropenia and anemia. Iron was given by mouth. Liver extract injections were made January 30, 31, February 4, 7, and 8. The temperature descended to normal in two days and remained within normal limits for five days. During this time, she consumed small amounts of liquids and a few soft foods, played with toys, noted her surroundings and in general seemed contented. Her face, however, was pasty, pallid, and puffy, the eyelids swollen. On February 5, the white count was 3,350, the red count and Hgb. unchanged. February 6, her temperature suddenly shot up to 104.4 (r) and the patient appeared extremely toxic. The swelling of the face and bloated appearance of the body increased. The sub-mandibular mass increased to the size of a lemon, was very tender, firm, and fixed. A mass in the left temporo-parietal region of the scalp which seemed fixed to the underlying periosteum and bone became more pronounced. A point of interest in this connection is that not only was this enlargement present at the very onset of illness in December but it had resulted from

a blow sustained in falling about three weeks before and had not subsided in the interval. In retrospect, after the diagnosis of acute leukemia by blood study was unmistakable, this incident had a significance which at the beginning I failed to appreciate.

The patient continued to lose ground rapidly. She was listless, apathetic, and evinced no interest in her surroundings. She did not play and took only sips of fluid when urged. Her temperature remained high and the pulse ran at 140 to 150, fair in quality at times but most often poor. February 8, because of an obstructive edema of the throat, she could swallow nothing. The floor of the mouth was swollen to such an extent that the mucous membrane, pale, watery, and distended, protruded over the lower incisors between them and the tongue. There was no bleeding from mucous membranes nor had there been at any time previously during her illness. February 9, a transfusion of 200 c.c. of citrated blood was given into a right antecubital vein. About eighteen hours after the transfusion, she was able to swallow liquids again. The temperature slowly fell to 101 by February 11, and the patient improved somewhat clinically. She was more contented, less distressed and irritable. The prognosis was of course as hopeless as ever and the child was discharged from the hospital when the father decided that if she must die she might just as well die at home.

February 13, two days after discharge from the hospital, her axillary temperature was 105 and the white count 6,000. Swelling of left cervical nodes rapidly increased. Inguinal and axillary nodes were palpable but small. At no time did these nodes become larger. Blood smears were submitted on this day and the report showed no more immature leukocytes but the polymorphonuclears showed severe toxic changes. The differential count was: polymorphonuclears 68 per cent, lymphocytes 30 per cent, monocytes 2 per cent. By February 19, the mass under the left jaw was fluctuating. It was incised and thick pus was freely evacuated. By February 22, her general appearance and condition were good. The patient was rapidly recovering strength, appetite, and normal activity. On February 25, the white count was 6,000, the blood smear report showed great improvement: no immature leukocytes and polymorphonuclears no longer appeared toxic. In the differential the polymorphonuclears were 58 per cent, lymphocytes 36 per cent, monocytes 6 per cent. Thus, during the following month, the patient was to all appearances and from all evidence a normal, healthy child.

On March 30, the father informed me that the patient had been worse again for the past week because of a "cold." On examination the child presented an appearance of marked toxemia and prostration. The temperature was high, the pulse weak and rapid, and the skin was literally covered by firm gray-blue nodules varying in size from that of a match head to that of a walnut, the largest infiltrated area being in the left temporo-parietal region. This mass, although less discolored than the remainder, had begun to assume a cyanotic hue. On close questioning the parents recalled that during the first week in March several of these nodules had been noted below the left clavicle, but inasmuch as they were small and painless and no more of them developed I was not advised of the circumstance. Furthermore, the child seemed well and happy. Shortly after her "cold" began, however, many more nodules rapidly appeared. The white count was 160,000; Hgb. 60 per cent. Examination of the chest revealed a large area of dullness suggesting pneumonic consolidation in the anterior portion of the left lower lobe. Because of abdominal distention, enlargement of the liver and spleen could not be definitely determined. There was no ulceration in the throat nor was there any evidence of bleeding from mucous membranes. A blood smear was submitted to the Department of Pathology, University of Minnesota, and again showed a typical acute lymphatic leukemia, nearly all of the white cells being immature lymphocytes. The child appeared moribund and the parents were so advised. General symptomatic measures of therapy were instituted and for the lack of anything better to do an injection of liver extract was given into the buttocks.

Due to the severity of the weather conditions and the comparative inaccessibility of the farmhouse, the child was not seen again until five days later, at which time examination revealed that the cutaneous nodules had all, to a marked extent, faded in color and diminished in size. The large temporo-parietal mass had decreased to one-third of its previous size. The other scalp nodules were scarcely palpable, whereas five days before they had been very obvious to sight and touch. The area of dulness in the chest had diminished. Clinically, she had definitely improved. Her eyes were brighter, she took an interest in her surroundings, and talked a little for the first time in a week. The temperature and pulse, however, remained rather high and the white count was 140,000. The possibility of liver extract playing a rôle in the child's improvement seems too remote for consideration for, of course, in all probability its use was coincidental with an attempted reversion to normal on the part of the hematopoietic mechanism. Yet, it is conceivable that some factor or factors in the body may be essential to prevent an uncontrolled over-production of certain blood cells just as the factor which is known to be present in liver extract is essential to initiate and sustain a state of normality in red cells where, one might say, under-production exists. The premise of a system of checks and balances in a blood cell controlling mechanism or mechanisms, which, in the normally functioning human body, prevents a shift toward abnormality in either direction is not without logic. There being at hand no other preparation which might conceivably provide whatever it was, if anything, that was lacking in this instance, liver extract was selected. Thus, I tried to justify submitting this already unhappy child to the additional discomfort of liver extract injections.

From about April 1 to April 9, clinical improvement was steady, being most marked the first five days following March 30. The nodules continued to disappear, although more slowly. Nodules known to exist one day would have disappeared entirely the next. The large temporo-parietal mass became hardly palpable. The

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MEDICINE IN WASHINGTON AND CHISAGO COUNTIES

ONE of the upper tributaries of the Mississippi is the St. Croix River, which forms part of the boundary between Wisconsin and Minnesota. It is also the eastern boundary of Washington and Chisago Counties. In the past it was the only approach to this region. Explorers, voyagers, and missionaries traveled its waters as it was one of the routes between the Mississippi and Lake Superior.

Until the treaties of 1837 with the Sioux and Ojibway Indians, there were few, if any, white settlers along its banks. White men knew the valley as a place of danger and the Indians referred to it by the ominous name of the "Valley of Bones," due, no doubt, to the frequent encounters then between the two hostile tribes.

With the signing of the treaty, the St. Croix valley was open for settlement. Lumber was the incentive which attracted the white man, and with the coming of the lumberman, medical history of the region begins. Among the first to arrive was a Dr. Fitch. In 1837 he and a group of others, including Franklin Steele, Jeremiah Russell, and a Mr. Maginnis, came from Fort Snelling and built a rough cabin near the falls. They then separated to search for pine, Dr. Fitch exploring the Sunrise River. A year later Dr. Fitch returned in association with the St. Croix Lumber Company. No other information about him is available except that he is said to have come from Galena, Illinois. Scarcely more is known about a Dr. Green, who came up the river in the fall of 1838, also looking for lumber. Green and his companions returned the following spring with complete materials for a mill, and on April 27, 1839, they drove in their stakes and formed at Marine the first permanent settlement in Minnesota. The mill was completed and at work by fall. Green remained at least two years, cutting timber near Kettle River. It is doubtful whether either Fitch or Green practiced; if they did it was probably incidental, for it was evident that their prime interest was lumber. This doubt is verified by the fact that all the early records speak of Dr. Christopher Carli, who came in 1841, as the first practicing physician north of Prairie du Chien. Dr. Carli's history is more complete than that of his predecessors.

Dr. Carli was born at Frankfort-on-the-Main December 7, 1811. He was educated at Heidelberg University, where he studied medicine. In 1832 he came to this country and practiced for three years in Buffalo, New York, before returning to Germany. However, after two years he again came to America, this time spending a year in Chicago, then a year in New Orleans, then going back to Chicago again. On May 24, 1841, he settled in Dakotah, St. Croix valley, on the site that is now Stillwater. His practice extended from Lake Pepin to Lake Superior, and from Menominee, Wisconsin, westward. He was always ready to visit a patient, on foot, skates or snowshoes, by horse or birchbark canoe. He was a member of the first city council of Stillwater and officiated as city and county physician, and coroner. He opened the first bank in Stillwater, and started the first drug store, which he afterwards sold to Dr. Henry Murdock. He also opened a stone quarry, dealt in real estate, and had many other avocations, for, like most of the pioneer physicians, he

was obliged to rely on something besides his profession for his support. He lived in Stillwater until his death in 1887.

As more settlers came to the valley another physician, Philip Aldrich, arrived. He was born in 1792. There is no record of his early life or education. He came in the early forties and was mail carrier for the entire valley. At different times he brought mail from Prairie du Chien and Fort Snelling, going as far as the falls and treating patients along the route on every bimonthly visit. Whether he had ever earned the title of Doctor or not, he performed the duties of physician wherever he was able. He was a prominent citizen and occupied many public offices, including judge of probate and justice of the peace. He donated land for the town of Buena Vista. Although a Wisconsin man, he was well known and active on the Minnesota side of the river.

The next newcomer, Dr. J. M. Covey, also settled at Stillwater in 1844. He was a regular physician, having received a medical education in his native state of New York. He is described as being a sociable but eccentric man, and slightly deaf. He died in 1851 supposedly from the effects of sleeping in a newly plastered room. In 1846 and 1847 two more physicians arrived about whom practically nothing is known. The first was a Dr. De Witt, who located at St. Croix Falls, and the other a Mrs. Page, a practitioner of the Thompsonian school at Hudson. At the close of the Mexican war came Dr. Otis Hoyt, who stayed at St. Croix Falls until 1851, practicing medicine and "delivering pleasing lectures on the Mexican war," in which he had served. He also practiced a year in Stillwater, then moved to Hudson, where he remained until his death in 1885. He was the most prominent physician in Western Wisconsin for many years.

One other physician arrived in the forties, a Dr. Wright, who settled at Marine in 1849. Nothing is known about him except that he died there in 1851. It is interesting to know that he was reported to have died of cholera. Since Asiatic cholera occurred at Stillwater about that time, it is possible that it also reached Marine, brought by the river boats. Dr. Wright is referred to as the first physician at Marine, which adds to the belief that Dr. Green never practiced there.

During the next decade a tremendous number of immigrants poured into the valley, and with them the necessity for more medical care increased. Epidemic and other diseases, new babies, the treacherous current of the river, the knives that were freely drawn in the saloons, and the frequent accidents with the dangerous circular saws of the lumber mills conspired to provide a steady stream of patients, and doctors arrived in rapid succession to fill the need. Drs. Pugsley and Ahl came to Stillwater in 1850, and in the years from 1854 to 1857 they were joined by Drs. Murdock, Noyes, Muller and Rhodes. Marine became the home of Drs. Cooley, Reiner and Gaskill, and in 1854 Dr. L. B. Smith, the first resident physician of Chisago County, settled at Taylors Falls. Smith had spent a year in St. Croix Falls (the town directly across the river, in Wisconsin), where even on the day of his arrival he found a patient waiting for him, a man who had been caught in a running saw, and whose arm was badly mangled. Amputation was necessary, but unfortunately the baggage which contained the doctor's larger instruments had not arrived, having been sent on a slower steamer. The operation was, therefore, performed as well as possible with a few small pocket instruments and a crude joiner's saw, and it is recorded that the patient recovered quickly. Smith was followed in 1855 by Dr. E. D. Whiting, who many years later married the widow of Dr. Smith.

Also in 1855 came Dr. E. W. Johnson of Lakeland, and Dr. J. W. Comfort of Wyoming. All of these physicians remained for many years and were prominent in their profession as well as in other activities.

Dr. H. W. Murdock's drug store, which he purchased in 1854 from Dr. Carli, appears to have been a busy place at this time, for many advertisements appear in the *St. Croix Union*, the only newspaper published in Stillwater at that time. For example, the following appeared first on February 13, 1855:

PILLS, PILLS

Pills old and young, of all qualities, kinds and descriptions. Enough to physic the territory at

Murdock's City Drug Store

Later, the doctor added a full line of perfumery, patent medicines, pure wines and liquors "for med. or mech. purposes," cigars, a soda fountain, paper hangings, oil paints and dye stuffs. He took as his partner Dr. O. G. Babcock, who came in 1855. Babcock moved to Afton in 1858, remaining there until his death in the fall of 1870. Carli's many activities are suggested by advertisements in the *Union*. On August 25, 1855, he advertised a hotel for rent, and offered for sale hardwood, 50 wheelbarrows, a two-horse wagon, 250 barrels of lime, 1,000 bushels of corn, 250 bushels of oats and 50 barrels of flour. Hoyt advertised land for sale. Crandall and Babcock also had a drug store, as did Pugsley and Reiner. These latter two were partners in practice at that time, as were Carli and Noyes. Later both partnerships were dissolved. In 1858 Pugsley formed a partnership with a newcomer, a Dr. Harlow. This partnership also was discontinued after several months. Apparently it was the custom for a newcomer to associate himself with a physician already established until he became known to the community.

In politics, in the forming of towns, and in the civic activities of their communities, many of the physicians played a prominent rôle. Reiner was a state senator; Gaskill a member of the first legislature. Reiner and several others served on the school board. Carli and Murdock helped organize the Stillwater fire department. Gaskill served three years as supervisor of the town of Marine. Later he was elected councilman, then elected and re-elected president of the council, and when Marine became a village he helped draft the charter. He was also a trustee of the state prison. Whiting was a representative in the legislature, and Carli was chosen president of the Old Settlers Association. Nearly all the doctors served as county, city or prison physician.

There is little information about diseases and epidemics in the earlier years of the settlements. The usual troubles were all present: tuberculosis, typhoid, scarlet fever and diphtheria. Asiatic cholera also reached Stillwater, carried there by the river steamers. Two interesting interviews with oldtimers are worth quoting in this connection. The first, Captain Stephen Hanks, of Albany, Illinois, remembered the cholera quite well:

"In 1851 or 1853," he said, "when I was rafting logs from the St. Croix to St. Louis, four of my men died of cholera on the way down. Two were taken ill in the evening and were dead in the morning. We stopped at the sand point between Willow River and Catfish Bar and I furnished the wood for coffins and Bowles made them. We buried them about a mile and a half above Catfish Bar on the top of the bluff. The rest of my crew deserted at Willow River. . . ."

The other interview, with E. W. Durant of Stillwater, included the following information:

"When the cholera first appeared on the river we were much afraid of it, but later, although it became very common, we paid little attention to it. The cholera came in 1849 to 1851, and spread from New Orleans up the river. All boats carried cheap board coffins and buried those who died when they stopped to take on wood, at night, if possible. . . . Another time, on the steamer "Cora" on the way up we buried several and at Stillwater landed a family of whom several died. Again, I attended two men on a log raft at Stillwater. I saw them in the evening and called again in the morning; one man at that time had lost half his weight and was very filthy, lying in a pool of mucus and bowel discharge. He died shortly after sunrise. I think four or five of that family died on the same trip up. We landed a cholera patient at Prescott, but I saved him; I used to give them Perry Davis Pain Killer internally and rub it on externally as well."

There is no way of estimating the extent of communicable diseases, because if any records were kept, they have since been lost. While the newspapers carried some death notices they rarely mentioned the cause of the death, and even when they did it was often such a vague term as "inflammation" or "complication" or merely "fever." Not all deaths were reported. This may have been due to the lack of news value in most cases, but it was evidently also due, in part, to a sense of civic pride which led the editors of the papers to deny any lack of health in their own communities. Epidemics were often reported by the papers of another town, but denied by the citizens of the town in which the epidemic was reported. It is hard to tell which was correct. However, deaths of a more sensational nature—fights and accidents—were often recorded. The latter were amazingly frequent, especially in the sawmills. Probably the mills had no safety devices then such as they have now, or possibly the men were careless; certainly the fast-running saws made an appreciable cut in the life and health of the community. One physician alone, in later years, reported five such injuries in three weeks.

The close of the decade of the fifties found the following physicians practicing in Washington and Chisago Counties: in Stillwater, Ahl, Carli, Dyson (a homeopath who came in 1858), Harlow, Muller, Noyes, Pugsley and Rhodes; in Marine, Cooley, Gaskill and Reiner, although the latter had spent two years in Stillwater; in Taylors Falls, Smith and Whiting, and at Lakeland, Dr. E. Johnson.

The next few years of medical history were much concerned with the Civil War, for many practitioners left to accept military positions. Some were killed; some settled elsewhere when the war ended; some stayed in the army. In 1861 Dr. Muller took an appointment as surgeon at Fort Ridgley. In April, 1862, Dr. Reiner was sent to Kentucky by Governor Ramsey to help care for the wounded. Dr. Reiner returned in May with "encouraging accounts."

The *Stillwater Messenger* for September 2, 1862, published the announcement that Dr. E. G. Pugsley was to leave, having been appointed assistant surgeon of the First Minnesota. On May 5, 1863, Dr. C. P. Garlick was appointed assistant surgeon of the Fourteenth Wisconsin Regiment, and in July, Dr. L. B. Smith was appointed an assistant surgeon, and left in April, 1864. Dr. Rhodes, in July of that year, obtained a situation in the Post Hospital at Memphis. He later became surgeon in a Minnesota regiment.

In the *Stillwater Messenger* for October 11, 1864, a note appeared that E. G. Pugsley, who had returned honorably discharged in February, 1864, had become assistant suregon in the Ninth Regiment. Pugsley built up an extensive practice in Glasgow, Missouri, though he never went back there after the war. Dr. L. B. Smith of Taylors Falls was killed the day before the battle of Tupelo, the division to which he belonged having been ambushed by Forest's troops. Smith

"was a tall man of fine personal appearance, with the air of an officer, for which reason, doubtless, some sharpshooter singled him out for destruction."

A Dr. H. M. Patterson died, although the war was only indirectly the cause of his death. The *Stillwater Messenger* for February 12, 1868, carried the following announcement:

"Dr. H. M. Patterson died on Sunday morning, a victim of a disease contracted during the late 'cruel war.' A boy in appearance, he was a man in soul. We knew him for years in the military, and during the past eighteen months we have met him almost daily in the civil walks of life. He was a promising young man of twenty-six years, proficient in his profession and the soul of honor and generous impulses."

There is practically no other information about Patterson—when he came or where he practiced.

Dr. Muller apparently never returned to practice in Stillwater, though he visited the town occasionally. Most of the other physicians did return, however, to the valley.

Besides serving in the army at this time, the physicians took an active part in both local and state affairs. At a meeting of the board of county commissioners in September, 1862, Gaskill was appointed county examiner of school teachers in the first district. In February, 1863, Noyes was appointed prison physician. Drs. J. B. Phillips and E. D. Whiting were mentioned as being at a legislative district convention at Marine in September, 1866. In April, 1868, there was a notice in the Stillwater paper by J. R. M. Gaskill, district clerk, and in August and October the *Taylors Falls Reporter* announced that Drs. Marshall and Griswold had been appointed examining surgeons for pensions. The next year some mention is made of "Senator Dr. Noyes." Whiting served in two sessions of the Minnesota state legislature (1860-1861) as a representative. Rhodes was also very prominent in civic affairs at this time.

Political activities were not the only ones in which the doctors took part. Christopher Carli advertised himself as a banker and broker. Ahl ran a hotel. Whiting made an extensive trip through Europe in 1867, visiting the Paris exposition and stopping at all the principal cities of the continent. In 1869 Rhodes invented and patented a very useful "spark-catcher" for the draft of parlor stoves. The same year a young men's course of lectures was planned, among which was one by Dr. A. J. Stone on "The Physical Education of the Coming Man," and one by Dr. Reiner on education. Dr. Kinkle was to have spoken, but was too busy at the last moment. Dr. Stone at this time issued a prospectus for a medical and surgical journal which appeared in Saint Paul a year later, in June, 1870. It was a monthly magazine called *The Northwestern Medical and Surgical Journal.* Stone was both editor and proprietor. Three Stillwater physicians were among those who, in 1869, formed the State Medical Society; they were Rhodes, Noyes, and Stone.

Among the newcomers of this decade were several very reputable practitioners as well as many disreputable ones. In 1860 came Dr. Hiram Murdock, the father of Dr. H. M. Murdock, who had been practicing in the valley for several years, and of Dr. G. W. Murdock who came to Taylors Falls in 1865. Hiram Murdock originally practised at Stillwater, but moved to the Falls in 1862, and stayed there until his death four years later. In 1861 Dr. G. M. Lambert came to Stillwater and in 1862 Dr. George Taylor to Point Douglas. In 1862 we find the following newspaper item:

EYES, EYES, EYES!

> Dr. J. A. Gilison, operator on the eye, will make a short stay in Stillwater at the Sawyer House . . .

Gilison highly recommended himself, offered to cure any ailment presented, and advertised "no charge for examination or opinion." He returned frequently to Stillwater and was typical of a large group of doctors who traveled from town to town, leaving behind a trail of useless medicines and valueless advice. In 1865 came Dr. C. A. Brooks, formerly an army surgeon, and the above mentioned Dr. G. W. Murdock. A peculiar and unfortunate event marked the arrival of Dr. Brooks. His first call, the day after his arrival in Stillwater, was to return to Minneapolis to attend his brother, who had been accidentally and fatally shot. In 1866 several physicians arrived. Drs. Marshall, Griswold and Spicer all came to Taylors Falls and settled there; also, the papers mentioned Dr. J. B. Phillips of Marine. In 1867 and 1868 a Dr. Carrington practised at Taylors Falls, and Dr. N. W. Beckwith, an eclectic, and Dr. H. Runge came to settle in Stillwater. Runge had a rather varied career. Born in Germany in 1817, he went to school in Russia, and graduated after a six-year course in the University of Berlin. He then studied to become a veterinary surgeon in Copenhagen for two years. After that he returned to Moscow and practised until 1863. Then he went to South America and later to Iowa, where he stayed until 1867, when he came to Stillwater. Later he spent eight years in Minneapolis, returned to Stillwater, and in 1880 settled permanently at Osceola. In 1868 Dr. Garnet spent the summer in Taylors Falls; and Dr. W. H. Cavin, formerly of Hudson, moved to Stillwater. In 1869 Drs. A. J. Stone, G. W. Hart, and J. C. Kinkle settled in Stillwater, and in 1870 Drs. Jordan, Morrow and A. J. Murdock began to practise in the valley, the latter at Taylors Falls. Some of these newcomers formed temporary partnerships with other physicians. Among these partnerships were Gaskill and Brooks (at Osceola), Kinkle and Noyes, Stone and Jordan, Spicer and Griswold.

With the coming of more physicians to the St. Croix valley, it was natural that some sort of a medical society be formed. There is no record of when the first one was formed, but in the *Stillwater Messenger* of May 27, 1862, the following brief announcement appeared:

> "The next quarterly meeting of the St. Croix Valley Medical Association will be held at Hudson on the 27th day of May, 1862, at 10 o'clock A.M. Surgical operations will be performed and advice given gratuitously for those who choose to present themselves before the association."

Evidently, then, there was some sort of medical society at the time, although there is no other record of its activities. It must have broken up later, probably due to the comings and goings of the physicians during the war, because in 1871 there were attempts to form a new organization.

The general health of the people in the counties during these years was poor, and epidemics were constantly being reported. Diphtheria was an ever present cause of worry; a large percentage of deaths reported in the papers were attributed to it. In March, 1863, the *Messenger* reported that "the dreadful disease prevails to a considerable extent in Hudson, having caused several deaths there." The same item recommended the use of ice, broken into small pieces and swallowed by the patient at intervals "until relief is experienced." Among others, one of Dr. Carli's daughters died of the disease in January of the following year. The Taylors Falls papers, in February, 1860, reported the disease prev-

alent, and stated that several children there had died of it. As to smallpox, in May, 1864, the Stillwater paper reported as follows:

> "We are reliably informed that this dreaded disease has made its appearance at Marine, having been brought to that place by a young man from Fort Snelling. When he was first taken down, it was not known to be smallpox, and many of the friends and neighbors called in to see him, thus spreading the contagion in an alarming manner. We have no further particulars. Our people would do well to take every precaution to guard against the disease as we can hardly hope to escape a visitation of the dreadful scourge."

Apparently it did not immediately spread to Stillwater, although early in 1870 it was said to be "all around." Ten or twelve cases were reported at the Falls, thirty or more in the vicinity of Prescott, and one light one, the first in eight or ten years, in Stillwater. The Taylors Falls papers reported seven cases in Osceola (on the Wisconsin side of the river), most of them mild, but one fatal. The schools there were closed because of it. The paper also quoted Dr. Carli as saying that it could be checked in a short time by vaccination, diet and quarantine. Apparently there was some attempt to isolate cases at this time, although it was not strict enough to stop any epidemic. Among other diseases, the chief ones mentioned were lung fever and hydrophobia. The *Messenger* of January, 1867, remarks: "Our physicians say that there is more sickness in this place and vicinity at this time than ever before known. The prevailing disease, which has assumed an epidemical type, is lung fever." Curiously enough there were actually no cases of hydrophobia reported, although there were constant complaints and much fear of unmuzzled dogs. One death from cholera was reported in August, 1863, a few miles from Stillwater, and measles and other less serious epidemics made their appearance.

Statistics concerning the diseases in the state prison at Stillwater at this time are available, and show fairly well the frequency of illness in that institution. In 1866 there were nearly two cases of sickness to every prisoner; in 1867 over five; in 1868 over six; and in 1870 over four. The number of convicts at the beginning of the year 1867 was only thirty-five; at the beginning of 1868, forty-five, and at the beginning of 1869, forty-two. Conditions in the prison gradually became better after 1868, there being fewer illnesses per prisoner, although of course more prisoners. On reading reports of the prison at this time one's greatest surprise will probably be that there was not more illness and a much higher mortality rate. While the food was generally agreed to be excellent, and some sanitary regulations were strictly enforced by the warden, nevertheless the ventilation, heating, toilet facilities and especially the drainage were very poor. Even several years later, when the Board of Health made its first report, the site was described as unfavorable; the soil about the prison was saturated by springs; stagnant water covered the prison yards at all times, making the lower walls damp. The lower tier of cells was untenable, having two inches of water standing on the floors, and the walls bulged noticeably and "might fall at any moment." The well in the yard was muddy from surface drainage. The privy was described as being in a filthy condition, and refuse from the kitchen was simply thrown out in the yard. The air was damp and musty, and the cells were ventilated only by four-inch openings into an air shaft, which most prisoners closed up in the winter because of the inadequacy of heating. The cells were only five by seven feet. Of course, the prisoners worked from six to six, with only a half hour for lunch, or, in winter, from five to seven, but they spent the rest of their time in these cells, eating and sleeping in them. The dark cells, for refractory prisoners, were "offensive and not properly ventilated," and the cus-

tom of chaining misbehaving convicts to the doors, in a standing position, prevailed. Besides these, later reports by prison physicians and inspectors reported bathing facilities most primitive, laundry arrangements poor, and the kitchen very poor, though the food was good. The hospital was inadequate and in many cases sick prisoners had to be treated in their cells. In 1876 the prison physician was still urging that a sewer be built, as there had never been even the most primitive one. He urged also that a competent medical graduate be employed as hospital steward, as the position was then filled by an incompetent convict. In 1877 the prison was so crowded that some of the five by seven cells held two convicts. It would be highly unfair, however, to blame prison officials for the poor conditions, as they were simply an echo of conditions in most of the towns at that time. Stillwater itself was far from modern in sanitary ideals. The average person at that time was quite ignorant, and new ideas were probably hard to introduce. For example, in the *Taylors Falls Reporter* in the spring of 1866, there appeared an excellent article on trichina, with illustrations, by a Chicago physician. In the following issue of the *Reporter* appeared a long comment on "the atrocious trichina story," saying what a hoax and fraud the former article was, that trichinosis existed only in a certain small part of Germany, where pork was eaten raw and uncured, and it was caused by the diet of the hogs, which ate snakes and vermin. The article assured its readers that trichinosis was never found in the corn-fed hogs of the surrounding territory.

When the decade closed, the following physicians were in the two counties: Ahl, Brooks, Carli, Gaskill, Griswold, Kinkle, Lambert, Marshall, Morrow, A. J. Murdock, Noyes, Reiner, Rhodes, Runge, Spicer, Stone and Whiting. Probably Drs. Beckwith, Cavin and Dyson should be included, although there is no further record of either their presence or their departures. Babcock and Jordan had both died in 1870; Johnson left in 1864, Geo. W. Murdock in 1868, and Hart in 1870. Muller and Pugsley both settled elsewhere after the war; Smith was killed, and Taylor of Point Douglas left after a year. The *Reporter* of February, 1870, said that there were only eleven licensed physicians in the district, which included Washington, Chisago, Pine and Kanabec Counties. In Washington County, in 1869, according to the report of the committee of credentials in the State Medical Society, there were four regular and five irregular practitioners.

(To be continued in August issue)

EDITORIAL

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BUSINESS MANAGER
J. R. BRUCE

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Clinico-Pathological Conferences in Hospitals

THE Council on Medical Education and Hospitals of the American Medical Association is charged with the duty of the certification of hospitals for the training of interns and resident physicians. This is what it says about one feature of its requirements:

"Necropsy performance in hospitals has rightly become a criterion of the scientific attitude of the staff. It reflects a desire to elevate the practice of medicine, an eagerness for scientific accuracy and a recognition of pathology as a sound basis on which to build clinical knowledge. To interns and older physicians alike there is a steady accrual of knowledge through repeated postmortem studies *in which pathologic and clinical manifestations are carefully correlated.*"

"To comply with the requirements of the Council a hospital approved for intern training must maintain a necropsy rate of at least 15 per cent of its deaths in order to supply an adequate amount of teaching material for the instruction of the house staff. *Surgical as well as necropsy specimens can be used to advantage in clinical pathological conferences, which should be conducted weekly or biweekly in accordance with the amount of material available.*" (The italics are ours.)

(J.A.M.A. 110:974, March 26, 1938.)

Again, the American College of Surgeons is responsible for the accrediting of hospitals for standardization and here is clause (b), section 3 of its Minimum Standard for Hospitals:

"That the medical staff review and analyze at regular intervals their clinical experience in the various departments of the hospital, such as medicine, surgery, obstetrics and the other specialties; the medical records of patients, free and pay, to be the basis for such review and analysis."

(Am. Coll. of Surg. 1938 Year Book, p. 57.)

So much for what may be called the letter of the law. There are additional reasons why clinico-pathological conferences have become necessitous. In private or voluntary hospitals, for instance, the opportunities for teaching interns and giving them a chance to put what knowledge they have acquired into practical application are very much less than in the larger public institutions, consequently positions in the latter type of hospital are much more sought after and the private hospitals have difficulty in securing their interns. Having done so, however, are these private hospitals not obligated morally to make up, as far as they possibly can, the deficiencies in their teaching and clinical opportunities for their interns? Certainly they are, and in no better way can they do it than by carrying on well planned conferences. Every hospital has an abundance of material, in both the living and the necropsy fields, and it is the duty of every staff, we believe, to see that such material is made available and presented properly each week. If the hospital has a full-time pathologist who is capable of taking charge of it, so much the better, but sometimes the hospital's internal arrangements point to the wisdom of a different plan, with a well informed

clinician looking after it. This arrangement is in effect in the oldest and best known hospital clinico-pathological conference in Minnesota. At another hospital a very successful conference has been carried on for several years by the roentgenologist. In both instances the pathologist collaborates very capably. In all cases the interns should be required to attend regularly and their duties so arranged as to permit them to do so. They should participate in the work themselves to a measurable extent. In one hospital conference they are required to make up the abstracts of the cases to be presented and read these themselves, then the staff members who have been connected with the cases amplify the findings and discuss them, following which a general discussion is carried on.

In any hospital where a conference of this type has been successfully established we believe that the morale of the staff is improved. Free discussion of difficult clinical problems, either in living patients or from those which have come to necropsy leads to clearer understanding and greater confidence between its members and to the creation of a better atmosphere in all the hospital's activities.

Finally, and most important of all, the entire quality of professional service in a hospital where its clinical and pathological problems are attacked at close range by its staff in a body is benefited and elevated. To know that the details of any case in the hospital may be utilized at short notice for full and open discussion in these conferences stimulates effort on the part of any who may be concerned. Closer attention is given to the verification of entries in the record and omissions are made up; more care is taken in the history writing and not seldom is the literature consulted, if there is any probability of a case being presented in conference. The work of the interns is scanned more closely and they themselves are found to take keen interest in having their notations above criticism. They seem to enjoy these conferences, too, and make no effort to stay away from them, once they have learned their value. As for the staff, it is their opportunity. They can make these conferences agreeable, profitable occasions if they will. Invariably, experience shows that the busiest and best trained men on staffs are the most regular attendants. They contribute freely of their time and ability and get much in return. It is this that builds up and maintains the spirit of these undertakings and makes them indispensible.

G. C.

Vitamin K

IN 1935 Henrik Dam reported the isolation of a material, deficiency of which in the diets of chicks resulted in fatal hemorrhage. He found this fat-soluble material in pig liver fat and designated it vitamin K. Subsequently, this compound was prepared in concentrated form and recently has been crystallized by Almquist. Thus, in a short time, another apparently essential animal nutritive product has been recognized and some of its basic chemical properties determined.

So-called vitamin K apparently is distributed widely in nature. In the plant kingdom its distribution appears to be confined almost entirely to the photosynthetic portion of the plant and in this its distribution is radically different from that of provitamin A. Considerable amounts of vitamin K have been found in alfalfa, kale, spinach, dried carrot tops, chestnut leaves, tomatoes, oat sprouts, and many other plants. In addition, it is found in soy bean oil and in the unsaponifiable portion of dog liver fat and pig liver fat.

The vitamin can be prepared from fish meal, rice bran or casein after the ether extracted material has been allowed to putrify and then subsequently extracted with petroleum ether. Under these conditions it appears that bacteria may cause the synthesis of this material but just what organisms can accomplish this synthesis are not as yet definitely known. However, Almquist and his associates recently have isolated a "fish meal organism" which closely resembles Bacillus cereus and which contains, and is capable of producing in fish meal, considerable amounts of vitamin K. The vitamin also is present in the droppings of chicks, in dried normal acholic feces of man, in dried colon bacilli, Bacillus subtilis, Staphylococcus aureus and many other microörganisms. Presumably the vitamin is present in the lipoid fraction of these bacteria but further data will be necessary before it can be assumed that the vitamin K found in alfalfa is the same substance as that present in microörganisms.

The exact physical and chemical properties of vitamin K are at present incompletely known but some properties seem to be definitely established. Physically it is a colorless, crystalline, fat-soluble material which is stable in air at 120° C. but is rapidly destroyed by alkali or sunlight. The substance apparently has a high molecular weight and contains a small amount of nitrogen but no sulfur or phosphorus.

Among young chicks which are given diets deficient in vitamin K there develops a tendency to bleed which is somewhat in proportion to the deficiency of prothrombin in the circulating blood. The syndrome in chicks is characterized by bleeding from the pin feathers and hemorrhages into muscles under the skin and in the abdomen, together with dark erosions on the lining of the gizzard. Frequently, low levels of hemoglobin are present and not infrequently fatal hemorrhages may develop very suddenly. Administration to these chicks of materials which contain vitamin K results in cessation of bleeding and an accompanying rise in the level of prothrombin. These phenomena are the basis for all known biologic assays of vitamin K.

The available experimental data are too meager to justify offering an explanation of the mechanism by which vitamin K affects prothrombin. Although prothrombin, so far as is known, does not contain a lipoid component, the possibility is not excluded that vitamin K, or a derivative thereof, might be present as a prosthetic group which is held in firm combination with the rest of the prothrombin molecule.

The recent demonstration that in the circulating blood of individuals who have jaundice there is a deficiency of prothrombin, has led to the clinical use of vitamin K as a therapeutic aid in the prevention and control of this abnormal bleeding. Independently, several groups of investigators have reported the successful elevation of the prothrombin level and in some instances inhibitory effects on actual bleeding of individuals with jaundice.

These results give some hope for control of the hemorrhagic diathesis in jaundice. However, simpler methods for estimation of the level of prothrombin in the blood and a more easily available and at the same time nearly chemically pure source of vitamin K are much needed at present. The recent successful use of injectable concentrates of vitamin K gives hope of standard dosage and of a standard method of administration.

H. R. B.

The A.M.A. Meeting

THE eighty-ninth annual session of the American Medical Association at San Francisco was outstanding in many respects. The attendance was large considering the location, approximately 6,000 registrants. As usual in recent years, much time was spent by the House of Delegates on economic questions. The president, Dr. J. H. J. Upham, in his address referred to the multiplicity of medical meetings and to the increasing growth in many localities of postgraduate courses, the demand for which appeared to be growing. He depreciated the lay control of scientific medicine and favored the campaigns on cancer, tuberculosis, and syphilis.

Dr. Irvin Abell, president-elect, was most enthusiastic over the excellent type of medical service now rendered to the American people, comparing the low morbidity and mortality here with that of other countries. He estimated that medical service rendered to the poor by American physicians amounted to over a million dollars a day. He said there should be no compromise between the profession and lay groups in the manner in which that service should be rendered. He also favored graduate extension.

President Wilbur of Stanford niversity stressed the need for more and better instruction for graduate and undergraduate students and suggested a three year pre-medical preparation for prospective students of medicine, adding that the quality of the applicant was of more importance than anything else.

Dr. Rudolph Matas of New Orleans was awarded the distinguished service medal of the Association. This is bestowed for outstanding achievement in Medicine and Surgery each year. Dr. Rock Sleyster of Wisconsin is the new president-elect.

Dr. H. H. Shoulders of Tennessee was elected Speaker of the House of Delegates succeeding Dr. Nathan B. Van Etten of New York.

Dr. Austin Hayden of Chicago and Dr. C. B. Wright of Minneapolis were each elected to succeed themselves as trustees for the ensuing five years.

The technical exhibits were many and varied. The scientific exhibits were generally considered very good but hardly the equal of the past few years.

Meeting places for the next three years were selected as follows: St Louis, Missouri—1939; New York City—1940; Cleveland, Ohio—1941.

The Duluth Meeting

THE annual meeting of the Minnesota State Medical Association held at Duluth, June 28 to July 1, was one of the most successful in the history of the Association. The program throughout was exceptionally interesting. Those in charge of the program were fortunate in obtaining a number of noted men from outside the state, among them being Dr. Howard W. Haggard of Yale University, and Dr. Irwin Abell, president of the American Medical Association.

Dr. George A. Earl of Saint Paul, the president-elect, will succeed Dr. J. M. Hayes at the end of the calendar year. Dr. J. C. Jacobs of Willmar was chosen first vice president, and Dr. A. M. Hanson of Faribault, second vice president. Dr. B. B. Souster of Saint Paul was elected secretary, succeeding Dr. E. A. Meyerding. Dr. W. H. Condit was re-elected treasurer.

Dr. H. Z. Giffin of Rochester was elected chairman of the Council. Dr. L. L. Sogge of Windom was re-elected to the Council from the Second District; Dr. B. S. Adams of Hibbing was re-elected from the Ninth District, and Dr. E. Mendelssohn Jones of Saint Paul was elected from the Fifth District, succeeding Dr. George A. Earl. Dr. W. A. Coventry of Duluth and Dr. W. F. Braasch of Rochester were re-elected delegates to the American Medical Association.

Dr. W. W. Will of Bertha was re-elected speaker of the House of Delegates. Dr. E. A. Meyerding, who has retired as secretary, was elected vice-speaker of the House of Delegates, and was presented with a gold medal for distinguished service to the Association over a period of many years.

The next annual meeting will be held in Minneapolis.

In Memoriam

Henning F. B. Wiese

1889-1938

DR. HENNING F. B. WIESE was born June 17, 1889 at Nordfjord, Norway, the son of Christian and Johanna Wiese. He was the youngest of seven children. His paternal great grandfather and grandfather were physicians in Norway. His maternal grandfather and one uncle also practiced medicine in Norway. His father was an expert accountant. One of his brothers is a physician in Norway and a sister is married to an English physician who resides in Paris, France.

Dr. Wiese attended the Gymnasium at Oslo, Norway, from 1904 to 1907, after which he entered the University of Oslo, graduating in 1915 with the highest honors of his class. He served his internship in the Rikshospitalet in Oslo, at the end of which time Dr. Mathiesen of Eau Claire, Wisconsin, who was Dr. Wiese's godfather, invited him to spend some time with him and Dr. Midelfart in their clinic, which he did, staying one and one-half years. From there he entered the Mayo Foundation at Rochester, Minnesota, where he spent three years. He was called by Dr. Schilling, the chief surgeon, to the Ullevaal Sykehuse in Oslo, Norway, at which hospital he served as surgeon for two and one-half years.

He received the degree of Master of Science in Surgery in 1922 from the University of Minnesota. He was a Fellow of the American College of Surgeons and the American Medical Association and a member of the Minneapolis Surgical Society. He was a member of the Kristiania Surgical Society and the Norwegian Medical Society; also a member of the Alumni Association of the Mayo Foundation.

During the World War Dr. Wiese held the commission of First Lieutenant in the Medical Reserve Corps and the heavy artillery of the Norwegian Army.

He gave his services for many years to the Wells Memorial Clinic and the Ebenezer Home. and he was a member of the Surgical Staffs of Asbury, Swedish, Fairview and Deaconess Hospitals.

Dr. Wiese was married on December 16, 1922, to Juanita Wood, daughter of Frederick and Fanny Wood of Eau Claire, Wisconsin. They have two children, Karin, age 14, and Stetson, age 11.

Above all else Dr. Wiese loved his family and his home, and it was his desire to remain there throughout his final illness, which lasted seven months. It was my privilege in association with Dr. Charles Hallberg to see him a great deal in the final weeks of his illness and to note the faithfulness with which his beautiful and talented wife cared for him. He was brave, patient and considerate of others to the end, which came at noon, Friday, April 29, 1938.

Dr. Wiese was one of the most ethical surgeons

(Continued on Page 528)

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
W. F. Braasch, M.D., Chairman

State Meeting

The Minnesota State Medical Association was holding its 85th Annual Meeting at the Hotel Duluth in Duluth as this issue of MINNESOTA MEDICINE went to press.

See the August issue for a complete résumé of events of this meeting.

AT SAN FRANCISCO

It is probable that there were more interested lay spectators in all parts of the country than ever before when the House of Delegates of the American Medical Association met in San Francisco in June, 1938.

Since the eventful session of 1937 in Atlantic City, the celebrated controversy, so-called, between the American Medical Association and the Committee of Physicians with their 430 signers broke into the headlines. Medical policies and plans—once "caviar to the general"—abruptly became front page news, along with strikes, mobbings, elections.

The floodlight of publicity seldom reflects facts with anything like a scientific accuracy. The rank and file of physicians saw themselves aligned there with that quaint survival of another day, the "stand patter," and were naturally astonished and resentful.

It was natural too, perhaps, that the proposal made some time ago by which the physicians should organize and go into the propaganda business for themselves should gather momentum as a result.

Better Counsel

Fortunately, better counsel prevailed at San Francisco. The American Medical Association will not organize a bureau after the pattern of meat packing interests, the soap interests, the patent medicine interests, the liquor interests.

The spectacle of a publicity bureau financed by doctors, complete with slogans, contact men, expense accounts and high powered advertising experts is unappetizing, still, to the majority of doctors. They have no skeleton to hide, no pork barrel to roll over. They want to take care of sick men to the best of their ability and they want to help well men to stay well. In the process, they wish to earn a sufficient income to provide for their families. Of the two objectives they are probably just a little more interested in taking care of the sick than they are in providing a livelihood—and that is a special distinction they hold over many other professional and business men.

Unnecessary Luxury

There seems to be very small reason for such men to indulge themselves in the modern luxury of a gigantic publicity bureau and advertising campaign.

The advantage to be gained, even in modern America, which operates to some extent through a nice balance, made up of horse trading and manipulation between pressure groups, would probably not be worth the effort. It might, in fact, produce a grave and unfortunate reaction and the expense involved would be terrific. In fact, the demand for funds for the new venture might well eat into the great services to the public for which doctors are justly proud—their machinery for protecting medical education, their great Councils, their public health education.

The resolution which rejected the proposal of three states was definite, though it suggested, by way of gesture, to those who clamored for a new relation to the public and press, that all spokesmen for organized medicine conduct themselves, in the Latin phrase, with "suaviter in modo" at the same time, they maintained the absolutely essential "fortitur in re." It passed without a dissenting vote.

No Split in the Ranks

Thus, the American Medical Association emerged from the storm and stress of another year without the split predicted in the headlines by the 430 episode; and it is still firmly established, as to public policy, upon the foundation of the famous ten principles.

One of the ten points was modified, it is interesting to note, to disapprove the inclusion of the special medical services such as pathological examinations, x-ray work and anesthesia in group hospitalization contracts and providing for removal of hospitals from the association's approved list where either the public or the profession is exploited.

The large number of hospital insurance proposals now being promoted publicly and privately made a specific clarification on these points essential and desirable.

For a Department of Health

The Federal Department of Public Welfare included in the recent government reorganization bill was condemned from the start by the House of Delegates. In its place, the delegates at San Francisco again approved a cabinet department of health which should be directed by a medical man as secretary.

It was appropriate and essential that the delegates should point out specifically that they—and not the special societies or individuals anywhere—are the truly qualified representatives of medical practitioners in America and that their actions in San Francisco reflect the policies and beliefs of organized medicine in the United States. Members of the house who are affiliated with other special societies were urged to work toward a closer coöperation between all medical groups in the country.

Survey

Dr. W. F. Braasch of Rochester, chairman of the Minnesota State Medical Association's Committee on Medical Economics and also chairman of the American Medical Association's advisory committee on the current nationwide survey of medical facilities, presented an encouraging report to the delegates. This survey will lay the first sound foundation for changes where they may be found necessary in the current system of handling medical care for the indigent or the low income groups, and will be complete within the year, it is hoped. It will constitute a great contribution on the part of American medicine to the social readjustments necessary to our time.

Members who visited the American Medical Association exhibit at Duluth found a complete progress report to date from Dr. R. G. Leland, director of the Bureau of Medical Economics of the American Medical Association, and in direct charge of the national survey.

Reëlection for another five years of Dr. C. B. Wright of Minneapolis to the Board of Trustees, of course, is gratifying to Minnesota.

ECONOMICALLY SPEAKING

[Monthly Editorial Prepared by The Medical Advisory Committee]

A survey of the present economic situation finds the business man generally viewing it with distrust. Everywhere dissatisfaction with today's set-up is apparent.

Many educators and parents are becoming resentful of the rather radical departure from the old fashioned teaching of the 4 R's and fundamental ABC's.

Ministers find a new alarm in the wandering away from the old time religion of our forefathers and a tendency in some nations to scoff at the teachings of the Church.

Lawyers view the promulgation of the totalitarian state with concern knowing that if the new tendency in thought is carried out, the law of the land may be that of the dictator.

Men of medicine see in the future possible abandonment of the patient-doctor relationship with all that it means, complete state controlled health service and its many possible dire results.

But your Medical Advisory Committee has noted with marked satisfaction during the last year a tendency for the approximate 2,500 members of our State Association to think more as a group than as individuals knowing that that which effects one effects all; that the malpractice cases are lessening in Minnesota, smaller verdicts are being obtained in courts, there are fewer professional testifiers in evidence. Our medical work is becoming more effective in the state, and we are better united with each other; therefore, the public is served better. Rural and urban lines are being eliminated.

Not Utopia as yet, but we are thankful for the few miles we have gained on the road to a better understanding. Your neighbor's trouble or

success reflects on you and should be your concern. A good neighborly spirit makes for contentment.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

Non-medical "Interne" Pleads Guilty to Practicing Medicine without a License

Re: State of Minnesota vs. Halsted.

On June 1, 1938, Hugh David Halsted, twenty-six years of age, entered a plea of guilty in the District Court at Minnneapolis, to an information charging him with practicing medicine without a license. Following a statement of facts to the Court, Judge Edward A. Montgomery, sentenced Halsted to one year in the Minneapolis Work House and placed him on probation for one year. On April 19, 1938, Halsted was tried before a military court at Fort Snelling on a charge of misrepresenting his rank as an officer and misrepresenting his qualifications as a physician. He pleaded guilty to both charges and was given a dismissal from the military service of the United States.

(Photo courtesy of Minneapolis Star)

The investigation conducted by the Minnesota State Board of Medical Examiners, which resulted in the filing of a complaint against Halsted by Mr. Brist on behalf of the Board, disclosed that Halsted was born September 29, 1911, at Milwaukee, Wisconsin. Before moving to Minneapolis, in the summer of 1929, Halsted resided in Chicago, and Chattanooga, Tennessee, graduating from high school in the latter place. From 1930 to 1933, inclusive, Halsted was a truck driver for the Harriet Laundry in Minneapolis. For a short period in 1934, Halsted worked as a shoe salesman for Dayton's Department Store. Along in the late summer and early fall of 1934, he spent some spare time at the Minneapolis General Hospital. Halsted stated that he always had a desire to be a physician and this prompted him to "hang around" the Minneapolis General Hospital. From October 10, 1934 to January 1, 1935, Halsted acted as an interne at Minneapolis General Hospital, according to a letter written by Dr. C. E. Remy then superintendent. From January 1, 1935, to June 25, 1935, Halsted was an interne at $50.00 per month at the Deaconess Hospital in Minneapolis. When inquiry was made at Deaconess as to why no investigation had been made of Halsted's credentials as a physician, the explanation was given that Halsted had previously been at Minneapolis General, and they assumed his credentials were in order. On July 5, 1935, after fraudulently representing himself as a physician and a graduate of the medical school at Northwestern University, and that he was licensed to practice medicine in Illinois, Halsted signed a contract to act as contract surgeon for the CCC Camps in Minnesota. No investigation was made at that time by those in charge to ascertain Halsted's qualifications. He was assigned to Camp No. 703 at Schroeder, Minnesota. In October of that year he was transferred to Allen, Minnesota. During the winter of 1935 and 1936 he was at Camp No. 724 at Bay, Minnesota. In the spring of 1936 he was transferred to Two Harbors, and subsequently to Rochester and Fort Snelling. In September, 1935, Halsted received a commission as a first lieutenant in the Medical Reserve Corps of the United States Army. This commission was obtained upon his fraudulent representations with respect to his medical training.

In September, 1937, Halsted "promoted" himself to a Captaincy in the Medical Reserve Corps. Following his marriage in 1936, Halsted was paid $262.00 per month by the Government. In February, 1938, inquiry was made concerning Halsted's qualifications as a physician which led to his court martial on April 19th. The only medical education that Halsted ever received was in the Extension Division of the University of Minnesota in the year 1930-1931, at which time he was enrolled in a class in General Inorganic Chemistry. In the school year 1931-1932, he was enrolled again in a similar extension course and the records indicate that he failed.

This case again emphasizes the necessity of more care being exercised by hospitals in Minnesota in the selection of their internes. Halsted violated the Medical Practice Act of Minnesota while he was at Minneapolis General, at the Deaconess Hospital and in the CCC Camps. At no time, during his career, did he have any right to represent himself as a physician and surgeon, nor to assume to act in that capacity. Judge Montgomery, in passing sentence, remarked that he did not understand how Halsted got by as long as he did. While he was at Fort Snelling, Halsted resided at 2284 Highland Parkway, St. Paul; at the time of his arrest he resided at 4039 Pillsbury Avenue, Minneapolis.

Minneapolis Drug Addict Sentenced to Ten-year Term for Obtaining Morphine by Misrepresentation

Re: State of Minnesota vs. Kathrine Burkhardt.

On June 8, 1938, Kathrine Burkhardt, 36 years of age, entered a plea of guilty to an information charging her with obtaining morphine by fraud, deceit, misrepresentation and subterfuge. She was sentenced by the Honorable Edward A. Montgomery, Judge of the District Court at Minneapolis, for a term of not to exceed 10 years in the Women's Reformatory at Shakopee.

The defendant, a drug addict, is the first person to be prosecuted under the Minnesota Uniform Narcotic Drug Act for obtaining morphine by fraud or misrepresentation. This law was passed in 1937 by the Minnesota Legislature and provides, among other things, as follows:

> "Sec. 18. Restrictions on obtaining drugs.
> (1) No person shall obtain or attempt to obtain a narcotic drug, or procure or attempt to procure the administration of a narcotic drug, (a) by fraud, deceit, misrepresentation, or subterfuge; or (b) by the forgery or alteration of a prescription or of any written order; or (c) by the concealment of a material fact; or (d) by the use of a false name or the giving of a false address.
> (2) Information communicated to a physician in an effort unlawfully to procure a narcotic drug, or unlawfully to procure the administration of any such drug, shall not be deemed a privileged communication."

The maximum penalty for a violation of the Act is 5 years imprisonment in a state penal institution. The defendant in this case received a 10-year sentence because she had two prior convictions for felonies in the United States District Court.

The Minnesota State Board of Medical Examiners was asked to coöperate with the Federal Bureau of Narcotics at Minneapolis in the prosecution of this case. The facts indicated that the defendant was an

addict and between March 26, 1938, and May 30, 1938, had obtained 19 prescriptions for a total of 233¼ grain morphine sulphate tablets from six Minneapolis physicians. She obtained these prescriptions by misrepresenting her physical condition and falsifying her name and her address. On being questioned by Judge Montgomery, the defendant stated that she was raised at Wabasha, Minnesota, and that her true maiden name was Katherine Mahoney; that her married name was Burkhardt and that she was divorced in 1926. She stated that she had been addicted to the use of morphine for the past 16 years. On being questioned by Judge Montgomery with respect to her physical condition she stated that she suffered from chronic asthma: that she had a tumor on her spine and that she suffered from adhesions following an operation. The defendant gave her true address as 1155-15th Avenue S. E., Minneapolis, where she resided with a sister.

The defendant used many aliases in obtaining these prescriptions, among them being the names of Barnes, Berg, Varnes, Fairchilds, Johnson and Peschkie. In 1933, the defendant was sentenced by Judge Nordbye in the Federal Court in Minneapolis, to a three year term in the Federal Industrial Institution for Women at Alderson, West Virginia. This sentence was imposed for a violation of the Harrison Narcotic Act. In 1936, the defendant was sentenced by Judge Joyce in the Federal Court at St. Paul for a 10 months term in the Minneapolis Work House for a similar offense The defendant also was in the Minneapolis Work House on four different occasions for vagrancy and once for the unlawful possession of morphine, in addition to the foregoing sentences in Federal Court.

The Minnesota State Board of Medical Examiners wishes to express its appreciation of the fine coöperation displayed in this case by the Federal Bureau of Narcotics in Minneapolis under the supervision of Mr. Harry D. Smith, and also the fine coöperation shown by Mr. Ed J. Goff, County Attorney, and his assistants Mr. Peter S. Neilson and Mr. Allen T. Rorem. The Medical Board believes that the enforcement of this particular provision of the Minnesota Uniform Narcotic Drug Law will greatly reduce the number of addicts who go about this state and attempt to procure morphine from one physician after another for the sole purpose of satisfying their craving for narcotics. The Medical Board wishes again to particularly caution the medical profession against furnishing or prescribing narcotics for these addicts. Most of them will not submit to a physical examination nor do they want to be hospitalized. The Medical Board feels that the imposition of a few more sentences like the one in this case, will go a long way toward solving this problem. There can be no question but what these persons need medical treatment and hospitalization, but it goes without saying that their addiction cannot be removed in a period of a few months.

ORALSULIN

No oral insulin preparation has been accepted by the Council on Pharmacy and Chemistry. Over eleven years ago *The Journal*, in discussing "Enterocap Oralsulin" (Dec. 4, 1926, p. 1935), pointed out the lack of evidence for the efficiency of orally administered preparations of insulin and pancreas. No new evidence has been found to necessitate a revision of the statement published at that time. Recently the federal authorities charged with the enforcement of the Food and Drug Act seized a shipment of Enterocap Oralsulin and declared the product adulterated and misbranded. According to the government report, examination showed that the preparation contained no insulin and that the labeling bore false and fraudulent representations regarding the curative or therapeutic effects of the product (Notice of Judgment 27373). No products of Lafayette Pharmacal, Inc., stand accepted by the Council on Pharmacy and Chemistry. (J.A.M.A., May 28, 1938, p. 1858.)

OF GENERAL INTEREST

Dr. Floyd F. Clark, who has practiced in Duluth for more than thirty years, is a candidate for county coroner.

* * *

Dr. Hamilton Montgomery of Rochester recently addressed the Chicago Dermatological Society, of which he is president.

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Dr. C. B. Wright of Minneapolis was reëlected a member of the Board of Trustees of the American Medical Association at the convention in San Francisco.

* * *

Dr. Jere W. Annis (Minn. '35), for the past three years a fellow in medicine at the Mayo Foundation, has located in Lakeland, Florida, where he will practice internal medicine.

* * *

Dr. William Musfelt of San Diego, California, has finished his graduate course at the U. S. Veterans facility of Minneapolis, and has been transferred to Camp Custer, Mich.

* * *

Dr. W. R. Kostick is now located in Fertile, with offices over the Fox Theatre. Dr. and Mrs. Kostick and baby daughter moved to Fertile from Robbinsdale, where the doctor had formerly practiced.

* * *

The bill which was pending before Congress providing for a new building for the Army Medical Library and Museum, of which editorial mention was made in the June number of MINNESOTA MEDICINE, was passed by Congress and the building provided for.

* * *

Dr. L. J. Leonard of Minneapolis, who recently filed as candidate for Hennepin County coroner, is one of the few men in the United States who is both a practicing attorney and physician, and the only one in the Northwest licensed to practice both medicine and law.

* * *

Dr. Robert Leighton, Jr., a graduate of the University of Minnesota, who served his interneship at Minneapolis General Hospital, on June 1 became associated in the practice of medicine and surgery with Dr. W. F. Cantwell of International Falls.

* * *

Dr. G. B. Cross and family have left Hills, where they have lived for seven years, and moved to Lakeville, where Dr. Cross will be affiliated with the hospital at Farmington, as well as taking care of the practice at Lakeville.

* * *

The marriage of Miss Rose Jessen of Saint Paul and Dr. Maurice C. Rousseau, son of Dr. and Mrs. Victor Rousseau of Maple Lake, took place on June 18. Dr. Rousseau is a graduate of the medical school of the University of Minnesota, and his bride is a graduate of the St. Joseph's School of Nursing.

Dr. R. A. Murray of Hibbing took over the practice of Dr. S. Z. Kerlan of Aitkin, the latter part of June. Dr. Murray has been associated with Dr. J. R. Manley of Duluth. Dr. and Mrs. Kerlan have left for California, where their son, Robert, will enter the University of California this fall.

* * *

Dr. F. J. Hirschboeck of Duluth, program chairman · Dr. Charles N. Hensel of Saint Paul, president; and Dr. M. H. Hoffman of Saint Paul, secretary-treasurer, were in charge of the program for the semi-annual meeting of the Minnesota Society of Internal Medicine held in Duluth on June 4. The meeting was attended by fifty medical men from all sections of the state.

* * *

Dr. Archie Nissen has been appointed Assistant Superintendent of the St. Peter Hospital, succeeding Dr. Ralph Rossen, who was recently named Superintendent of the Hastings State Hospital. Dr. Nissen is a graduate of the University of Minnesota School of Medicine, where he specialized in psychiatry, and later did graduate work. He has served for the past two years on the medical staff of the St. Peter Hospital.

* * *

Among other similar courses offered at various centers in the state during May, an all-day postgraduate course in the care of mothers and babies was given at Worthington. The course was arranged under the auspices of the State Department of Health, the University of Minnesota, and the Minnesota State Medical Association with Dr. A. J. Chesley, secretary of the department of health, and Dr. E. C. Hartley, director of the division of child hygiene, in charge. The faculty for the course included Dr. Rae T. LaVake, Dr. Samuel B. Solhaug, Dr. Edgar J. Huenekens and Dr. Irvine McQuarrie of Minneapolis. Such gatherings are financed by the social security funds granted for the purpose to the state department of health.

* * *

Attendants at the American Medical Association convention in San Francisco in June were Drs. W. F. Braasch, H. H. Bowing, J. F. Wier, B. E. Hempstead and F. J. Heck, C. F. Dixon, and Waltman Walters, all of Rochester, Minnesota.

Dr. Braasch is a member of the House of Delegates of the Association and took part in the program of the American Board of Urology. Dr. Bowing addressed the meeting of the Los Angeles Cancer Society, and spoke both before the meeting of the A.M.A. and the meeting of the American Radium Society. Dr. Heck spoke before the A.M.A. meeting and also at the meeting of the American Society of Clinical Pathologists. After speaking before the section on surgery of the A.M.A., Dr. Walters went on to Vancouver, B. C., to deliver a series of talks at the summer school of the Vancouver Medical Association.

REPORTS and ANNOUNCEMENTS

MEDICAL BROADCAST FOR JULY

The Minnesota State Medical Association Morning Health Service.

The Minnesota State Medical Association broadcasts weekly at 9:45 o'clock every Saturday morning over Station WCCO, Minneapolis and Saint Paul (810 kilocycles or 370.2 meters).

Speaker: William A. O'Brien, Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota. The program for the month will be as follows:

July 2—Artificial Respiration.
July 9—Body Temperature Regulations.
July 16—Summer Complaint.
July 23—Art of Relaxation.
July 30—Teething.

AMERICAN CONGRESS OF PHYSICAL THERAPY

An intensive course in physical therapy will be held September 7-10, 1938, at the Palmer House, Chicago, just preceding the 17th annual convention of the Congress of Physical Therapy. The course will consist of lectures, conferences, clinics and demonstrations arranged primarily for physicians but open to a limited number of technicians properly sponsored. Applications should be sent to the Congress headquarters, 30 N. Michigan Avenue, Chicago. Upon acceptance, a fee of $25.00 will be required.

EAST CENTRAL MINNESOTA SOCIETY

The East Central Minnesota Medical Society met at Pokegama Sanatorium at Pine City on May 27, 1938. Dr. Walter P. Gardner, Anoka, and Dr. Edward J. Coffman, Anoka, were accepted as transfers into this society.

Dinner was served and in the evening a scientific program was presented by guest speakers. Dr. O. H. Wangensteen discussed thoracic surgery and a discussion of his subject was presented by Dr. D. Greth Gardiner. Dr. R. E. Hultkrans spoke on "Chronic Low Back Pain" and Dr. Stewart Shimonek followed with a short discussion of this subject.

SCOTT-CARVER SOCIETY

At the annual meeting of the Scott-Carver Medical Society held at Mudbaden, June 14, Dr. C. A. Stewart, of Minneapolis, spoke on "Dietary and Nutritional Problems in Pediatrics."

The following officers were elected for the coming year:

President—Dr. Earl Crow, Arlington
Vice President—Dr. Harold Havel, Jordan
Secretary-Treasurer—Dr. B. F. Pearson, Shakopee
Delegate—Dr. C. F. Cervenka, New Prague
Alternate—Dr. J. C. Klein, Shakopee
Censor—Dr. Alvin Westerman, Montgomery

PROCEEDINGS of the MINNESOTA ACADEMY OF MEDICINE

Meeting of April 13, 1938

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, April 13, 1938. Dinner was served at 7 o'clock and the meeting was called to order at 8:15 by the president, Dr. R. T. LaVake.

There were fifty-seven members and two guests present.

Minutes of the March meeting were read and approved.

Dr. H. E. Hullsiek read the Necrology Committee's Memorial to Dr. John T. Rogers, and a motion was carried that it be spread upon the records of the Academy and a copy sent to the family.

JOHN THOMAS ROGERS*

1867-1938

The Civil War was in every sense a social revolution. Through the sweep of the conflict the planting aristocracy of the South was as completely ruined as were the clergy and the nobility during the French Revolution. The very economic foundations of the planting system, including slavery itself, were destroyed.

Even before the unrest associated with the freeing of the slaves, the South had at no time a static population, and to the migrating groups Kentucky had ever added her share. Beginning with the settlement by Daniel Boone in 1775, through its admission to statehood in 1792, and up to 1890, it had been first an outpost, and finally a point of departure for those seeking fortune elsewhere. By 1880 it was no longer frontier, but a great commonwealth with thousands of prosperous citizens. Still the blue-grass state annually sent out many who could not resist the beckoning will-o-the-wisp of fortune. The last great migration occurred between 1880 and 1890. This exodus was the result of the moral and material disintegration incident to the great war. In this decade 50,000 souls left the state which, during its history, furnished the rest of the Union with some twenty governors, many senators, attorney-generals, justices, and other high officials. It was at this time that the Rogers family left Kentucky for Minnesota.

William Edward Rogers was born at Cane Ridge, Bourbon County, Kentucky, on August 12, 1835. On June 10, 1857, he married Margaret Vernon, descendant of Jacob Spears, revolutionary soldier. The fifth of their eight children was John Thomas Rogers, born July 18, 1867, at Versailles, Kentucky.

There is nothing to indicate that John Rogers' childhood was in any way different from that of any son of a comfortably landed proprietor of the South. However, before he was far on his way to manhood, the aftermath of the great civil strife descended with a vengeance and, like so many others, William Rogers sought fortune elsewhere. It is not strange that in 1886 he chose Minnesota.

St. Paul was in its first exuberant flush of youth. The population was 110,000; it claimed 864 manufacturing establishments, while 18,000 persons and 142 trains passed through the union station daily. The Robert Street bridge and that to Dayton's Bluff were being built; 3,573 buildings of various types had been completed that year; and the city was served by thirty-five newspapers and periodicals. As a further mark of progress, it was noted that already some of the horse-cars had been powered with cables.

The Rogers settled in St. Paul after a brief sojourn in the Dakotas. Young John was left in Louisville to complete his studies at Transylvania University, after which he joined them. The first note of him in this hustling and rather awe-inspiring city of French, Germans, and Irish from down river, is a listing in the St. Paul directory for 1887. Rogers, John—clerk for W. E. Rogers, real estate, 42 Globe Building—boards with W. E. Rogers, Chatsworth & Owasco.* How long he was employed by his father is not definitely known, and for some time he clerked in a haberdashery on Robert Street. Both of these positions were temporary and it apparently was not until he met Dr. Charles A. Wheaton that plans for his future were crystallized. Neither is it known in what manner he met Dr. Wheaton, but it probably was as a patient since he was sent to Brainerd at the instigation of Dr. Wheaton, with a note to a friend of the doctor's, remaining in the north woods six weeks. It might easily have been otherwise, since his father's office and the doctor's were but a block apart.

In 1888 he is living with his family in St. Anthony Park, and is a student with the firm of Wheaton and MacLaren. Dr. Wheaton at this time lived at 351 Washington Avenue, facing Rice Park, some three blocks from his office. Coming to St. Paul in 1877, he was well established in practice and was one of the northwest's surgical leaders. Young John helped about the office, read in the doctor's extensive library, and made himself generally useful. He assisted at operations, slept in the office and, in the absence of telephones, answered the night-bell, notifying Dr. Wheaton and driving him on calls.

In 1881 the St. Paul Medical College had taken into its faculty certain Minneapolis men, had moved to Minneapolis, and became the Minnesota College Hospital. In 1885 the St. Paul members resigned and again started the St. Paul Medical School, while the Minnesota College Hospital reorganized and became the Minnesota Hospital College. In 1888 the St. Paul Medical School and the Minneapolis institution surrendered their charters and became the College of Medicine and Surgery of the University of Minnesota,

*Memorial to Dr. John T. Rogers, read at the regular meeting of the Minnesota Academy of Medicine, on April 13, 1938

*Now Fairmount Avenue.

with a faculty of twenty-nine and Perry Millard as dean. The course of lectures consisted of three years of six months each, and it was here that John Rogers began his medical education in the fall of 1888. Living in St. Anthony Park, he took the Great Northern train at Merriam Park each morning and rode to the 10th street bridge. Here he got off and crossed the river to the medical school which was on 6th street, across the street from the old St. Barnabas' hospital.

Now for the first time were felt the tremors of the impending social and economic upheavals that were to mark the end of an epoch, that of the end of the period of free land—the last of the frontier. The post-war deflation had set in and the Rogers family had not been long enough in the new surroundings to have become well-established in business. John's father and brother, Nat, were in the stock business at South St. Paul, but with little surplus money left for medical studies. His arrangement with Dr. Wheaton not only was an opportunity to observe the practice of medicine and surgery first-hand, but was important from an economic standpoint as well. The financial status of the young aristocrat from the south was at times a sore burden.

One of the many stories he told to the writer dealt with the time when, because of well-worn shoes, he found himself wellnigh walking on the ground. His chief, affluent and a smart dresser, had noted this and caused a pair to be made, ostensibly for himself, but actually to John's measure. He brought them to the youth apologetically, saying that they did not fit him, and wondered if the young man might wear them, since it seemed a shame to allow them to be wasted. To quote Dr. Rogers, "Doctor, I was a proud young fool, I was extremely humiliated. That night when I walked across the bridge on the way to dissect I dropped the shoes into the river. I borrowed two dollars from a classmate and bought a pair of very fine shoes with paper soles." He told Dr. Wheaton the others had been stolen. I have heard Dr. Rogers repeat this story many times, with considerable amusement. It is an evidence of the fierce pride which, tempered by experience and maturity, was so great an asset in later years, and which so often enabled him to hold fast to what he felt was right.

Assisting at operations both in and out of the city —these were the days of weekend excursions to smaller cities with hospital facilities, and included turkey-shoots, banquets, poker-games, and Sunday morning surgical field-days—answering the office night-bell, driving Dr. Wheaton, dissecting evenings, spending the summer of the final year at the City Hospital, thus went the three years of from 1888 to 1891.

In 1891 President Harrison's difficulties with restless labor were beginning. The populist party was organized, great talk went on of gold and silver, the slogan "16 to 1" was mouthed about, falling wheat prices brought ruin of thousands, and on the streets of Chicago a curious crowd watched a vehicle which rolled along with no horse attached to it. In the realm of medicine, Witzel was performing his first gastrostomy, Quincke introduced spinal puncture, Bier brought out his hyperemia, a general aseptic ritual for surgery was standardized, and for the first time Halsted announced the use of rubber gloves. With this year came the end of John Rogers' first struggle for a foothold in the practice of medicine. His hardest days were behind him. Licensed to practice on April 10, 1891, and graduated in June of the same year, he is now a full-fledged physician associated with two of the best known men in the city, Dr. Charles Wheaton and Dr. Archibald MacLaren. He has left the family home, probably to be nearer his work, and is living at the Seville Flats.

Those who know him are acquainted with his meticulous methods in the matter of records and correspondence. It is questionable whether John Rogers ever ended a day's work without having completed his records and attended to his correspondence. Thus it is not surprising to find in a small pocket diary (an advertisement by the Maltine Company), careful notes of his first patients. He graduated in June, 1891, and what is probably the record of his first private patient appears on the page for June 12th, as follows: Wm. Swab—bartender—incised wound of forearm made by dirty hatchet—treatment: wound scrubbed with 1/2000 bichloride—iodoform dusted on—wound sutured with catgut—no inflammation.

On July 16th occurs the following: Dutchman—porter in saloon—got in row with waitress who stabbed him in buttock with butcher-knife. Wound three inches deep, very painful, bleeding profusely. Treatment: irrigation with 1/1000 bichloride, one silkworm suture, iodoform gauze. Union by first intention.

Apparently his first operation was a tonsillectomy. On September 20th this appears: Morris J.—Tonsillectomy. Enlarged and frequently inflamed tonsils—both removed under cocaine—no pain—little hemorrhage—no attack several months afterwards. On January 5, 1892, a cervical repair, and in February a breast abscess. In April a fractured clavicle and in June a mastoid abscess, described as follows: Baby T., 6 months old. History unsatisfactory. Had been scarlet fever in home but baby had not taken it. Did well for two weeks when it began to bleed after a dressing. Local doctor called but it didn't succeed with acid—I saw child 48 hours afterward almost in collapse—still bleeding—applied compress—administered whiskey—hot applications to body—recovered.

And so the record goes, with careful notes, comments, his own explanations of failure to get desired results—on through 1892—an abscess of the femur, another mastoid, in October of that year the first of a remarkable—for that time—series of ectopic pregnancies, described with great detail and occupying two pages of small script, a repair of a cervix and perineum. On February 29th of this year, John Rogers joined the Ramsey County Medical Society.

At the age of 26 years, on April 4, 1894, he read his inaugural thesis before the Minnesota Academy of Medicine, entitled, "The Use of Wet Dressings and Poultices in Surgery." In this paper he cites his success with 1/3000 hot bichloride of mercury solution used as a wet dressing, as a substitute for the still-popular flaxseed poultice, which he condemns. He

states that a recent communication from one of the men at Johns Hopkins says that flaxseed poultices are still used in that institution in cases of stitch abscess. He ends by saying, "I believe the day is not far distant when adequate incisions will be small ones, large drainage tubes will be entirely dispensed with, and irrigations unnecessary." A bold statement for 1894!

By this time his case-book includes such operations as excision of the superior maxilla, extra-uterine gestation, repair of crushed foot, mastoid abscess, suture of tendons, amputation of leg, varicocele, pyosalpinx, curettage, appendicitis, hemorrhoids, pelvic abscess, varicose veins, hysterectomies, anal fistula, Dupuytran's contraction, compound fractures, hernia, amputation at the thigh, osteomyelitis, and others. A widely varied surgical practice for a young man four years after graduation.

In June, 1895, he was appointed Clinical Instructor in Diseases of Children at the University of Minnesota. On June 20th of that year he read before the Gynecological Section of the Minnesota State Medical Society the first of his papers on ectopic gestation, with a report of sixteen surgically-treated cases.

Considering that it was but six years since Tait had published his work on extra-uterine gestation, it is again an evidence of the young doctor's progressive spirit when he says he believes that this condition "could and should be diagnosed before rupture in a large majority of cases." He states that if the operation be in the hands of experts and the patient operated upon before rupture, the mortality should not be greater than 2 per cent. This number of cases was unusual for the time, and called forth a rebuke from a surgeon in an eastern clinic attended by Dr. Rogers. The young surgeon had taken part in the discussion following an operation for ectopic pregnancy, mentioning his experience and that of his colleagues in St. Paul. The eastern surgeon apparently felt that the young man from the raw middlewest was overstepping the bounds and remarked witheringly that if these doctors had sixteen such cases there must be very uncommon women in St. Paul. Dr. Rogers had the better of it. He took great glee in his reply, which was to the effect that the women in St. Paul were in no way unusual, but it was possible that the doctors were.

In 1897 he published a paper on the operative treatment of tumors of the upper jaw, and in May of that year was appointed Clinical Instructor in Surgery at the University of Minnesota. It was also in this year that he and Dr. A. B. Stewart of Owatonna were sent as delegates to the meeting of the American Medical Association in Philadelphia, one being certified to attend the International Medical Congress in Moscow. They left St. Paul in May, sailed on the City of Rome, arriving in Edinburgh on June 16th, after two or three days in Glasgow.

Here they took courses in practical pathology, morbid anatomy with Alexander Bruce, and had the run of the Surgeon's hall and the Royal Infirmary. In July they left Edinburgh on bicycles for Vienna by way of Dublin, London, Paris, Lausanne, Berlin and Prague, arriving after a six-week's trip. In Vienna they settled down for several months of study. They took courses in pathology, special dissection, general medicine, gynecology, and a course in blood disease with technic of staining specimens. Dr. Stewart says in a recent letter, "John was a diligent student and a delightful traveling companion. I learned much with him and from him."

Changes have occurred in the firm in the past three years. In 1897 it became Wheaton, Wheaton and Dennis, John Rogers having left January 1, 1897, to practice alone at 145 Lowry Building. Bob Wheaton having died suddenly in 1898, the firm became Wheaton, Rogers and Dennis.

In June of that year Dr. Rogers presented before the State Society a paper on contused wounds of the abdomen and their surgical treatment. Here he makes a plea for early recognition of intra-abdominal injuries resulting from contusions, emphasizing the point that this condition had not been adequately dealt with in the literature, which he says is "incomplete and unsatisfactory in its treatment of this condition." He hopes to create more interest in this type of serious injury, leading to further study and more frequent reports of cases, since, as he says, ". . . the advances in modern surgery have made it possible to explore the abdomen with impunity and almost with safety." In the year 1901 Dr. Rogers was elected President of the Minnesota Academy of Medicine.

It is now 1902. The Spanish-American war has come and gone with its scandals and profiteering, the beginning of the Roosevelt ascendency, and the beginning and end of the United States' short-lived fervor for foreign expansion. John Rogers is now associated with Dr. Wheaton, the firm name being Wheaton and Rogers. Reading papers at meetings, giving talks in and out of the city, participating in both development of the education and economic side of medicine as well as the purely scientific, he was adding rapidly, through ability as well as personality, to what had already become a large following. Two more papers on ectopic gestation, one reporting twenty cases, both plead for earlier diagnosis and proper treatment, showed that when John Rogers believed himself to be right the chips might fall where they would. He handled without gloves those who, he felt, through failure to grow with medical science were failing in their duty. A paper written on cancer of the breast, one read at the Academy on surgical cases, immobilization of the lower jaw, and one on tumors of the upper jaw and their operative treatment had appeared. The last mentioned was written six years after graduation, and reported several personal cases.

In 1903 the firm became Wheaton, Rogers and Gilfillan, with offices at 170 Lowry Arcade, and in 1905 Wheaton, Rogers and Colvin. During these years we find Dr. Rogers appearing before assemblies with papers on such subjects as tuberculosis of the kidney, differential diagnosis of gallstones, surgery of the bileducts, surgical treatment of the gallbladder, gunshot injuries of the brain, gastric ulcer, intussusception. One article, entitled, "The Health Instinct," appeared in *Popular Science Monthly*.

During this, the gilded age in American scientific,

economic and social development, with revolution occurring with disturbing frequency in all branches of thought, John Rogers was not only an interested onlooker but an enthusiastic participant. The progress in science, the frequency of innovation in medical and surgical circles, the swiftness with which yesterday's beliefs were cast aside for today's, were staggering, and taxed one to keep abreast. Dr. Rogers' writings through these years show not only an attempt to do so, but a reasonable success.

The volume of surgery done in those days by any one or a group of surgeons is worth noting. At the time under discussion more of the work of this group was done at St. Luke's Hospital than at other institutions. We know that it was not all confined to one institution, and also that there were the above-mentioned weekend excursions to smaller towns, with operations on Sundays. Yet, in one of these years, over a hundred patients were operated upon by this firm at St. Joseph's Hospital. During the years from 1880 to the early 1900's much of the surgery from Minnesota to the West coast was done in the Twin Cities, as is shown in the variety of the cases. No one city could have furnished certain rather uncommon types of cases in such numbers as the records show.

Dr. Wheaton, having retired by 1912, the firm became Rogers and Colvin. At this time the City Hospital staff was reorganized with Dr. Rogers as chief. When the American College of Surgeons was founded in 1913 he was one of the founders. In January of this year he became Assistant Professor of Surgery at the University of Minnesota; and, in 1914, he married Lillian Hallam Cooley, making a European trip as his honeymoon. In 1914 he was president of the Minnesota State Medical Association. In his presidential address he dealt with such subjects as the liquor question, medical licensure, legislation, medical defense and expert testimony, ending with a discussion of the then burning question of medical education and the Medical School. Here again, his remarks show a refusal to compromise with what he believed to be wrong. In 1915 the Rogers-Colvin partnership was dissolved and Harry Zimmermann entered Dr. Rogers' office as surgical assistant, the firm becoming Rogers and Zimmermann. In 1916 he was made Associate Professor of Surgery.

Through all these years, the seed of an idea was growing in the mind of one of St. Paul's pioneer business men, the development of which was to be of untold benefit to those less favored than himself. This man was Charles T. Miller, erstwhile clerk for J. J. Hill and one-time partner of W. F. Davidson in the steamboat business. It had long been Mr. Miller's wish that a hospital be built and named for the one whom he held in such high regard and esteem—John Rogers. Dr. Rogers refused to accede to this wish, insisting that the hospital be named for its donor. Mr. Miller died without having carried out his plan, but it was periodically discussed by his widow and Dr. Rogers. When she died several years later, she made a bequest of $1,400,000 for the building and equipping of a hospital, a board of trustees being named at the same time. The sole stipulation was that there were to be fifty free beds, with no distinction as to color, race or creed. Dr. Rogers was named chairman of the board.

In 1917 a site was purchased and plans drawn for a hospital of 150 beds. According to the estimates made at the time, this could be built for $400,000, leaving $1,000,000 for a maintenance endowment. In the months intervening between the planning and the actual time of construction, the war had forced building costs so high that it was seen that the building as planned could not be erected and equipped and the endowment left intact. It was thus decided to postpone the building to a more favorable time, and allow the fund to grow.

Meanwhile, we had entered the war. In the summer of 1918, Surgeon General Gorgas arrived in St. Paul on a tour of the country in search of hospital facilities for American soldiers soon to return from France. After examining the Miller Hospital plans he advised the Board that if they would increase the number of beds to 250 and complete the structure, the government would take it over for a term of years. Patriotism and expediency finally caused the Board to accept this offer. The contract was let, construction rushed, and by the fall of 1918 the building was well under way.

In November 1918 came the armistice. With neither warning nor explanation, a telegram arrived unqualifiedly cancelling the afore-mentioned offer. The Board now found itself in a difficult position. It was obvious that the increased size of the hospital, together with still high construction costs, would leave little of the endowment fund for maintenance. After months of effort and after finally believing their troubles to be over, the Board suddenly found themselves, through a combination of circumstances, in a worse situation than before. Insurance, fire-protection, interest, heating, and other costs were mounting alarmingly. Permission was finally obtained from the court to use the endowment fund for the completion of the building, which was finished in November, 1920.

In order that the hospital might open, it was necessary to borrow, for the purpose of equipment, approximately $150,000. This was accordingly done, and the hospital opened its doors to the first patient on December 1, 1920.

The Charles T. Miller Hospital was not named for John Rogers, but those of us who know him realize that it is as much a monument to the dogged persistence, untiring effort, and almost fanatic devotion to an ideal, of one who was carrying out the wishes of a friend, as though the institution were named for him. It is safe to say that the only time in his life that John Rogers incurred the displeasure of anyone, was during the planning and building of this hospital, and then only because he held out against any and all influences which in his mind might in any way obstruct the furtherance of a great project. Fortunately, it was but a short time before all ill-feeling toward Dr. Rogers and the Miller Hospital was dissipated. From the time of its inception to the time of his death, John Rogers made the Miller Hospital his one thought. His com-

plete success is shown, I believe, in the fact that in no year since its opening has the hospital cared for less than 1,000 free patients, reaching a high mark of 1,775 in 1937.

From the several months of conferences with his confrères who were eventually to be financially responsible for the hospital's opening, came the idea of an association to practice medicine in a group. From this grew the Miller Hospital Clinic which was organized in 1922 and continued for the practice of medicine and the training of younger men until it was dissolved in 1934.

In looking back over my years of association with Dr. Rogers, I should say there were, of his many good qualities, three that were outstanding. The one obvious to all was, of course, his great personal charm, which instilled in those meeting him for the first time a feeling of respect and trust. The second, and not so obvious, was his tremendous capacity for work, with apparently little effort. John Rogers had a powerful physique, one which most of his life enabled him to outdo physically not only his contemporaries, but often younger men. As a woodsman he was indefatigable. His hunting partners for thirty years will testify to this. It was well known to his associates that he could do twice as much work as most, see an astonishing number of patients, and at the same time give the impression of being quite at leisure. At any time he was ready and willing to help with another's problem, be he intern, nurse, doctor, or friend—for John Rogers was interested in people. And, lastly, he was honest.

Had Dr. Rogers neither prescription-pad nor scalpel, I believe he could still have accomplished more for ailing humanity than many physicians. He had the very rare and precious quality which enabled him to do for a patient the one thing that a patient wants done when he consults a doctor—that of being made to feel better. Meeting and talking to John Rogers had that effect. Samuel Bard said one hundred years ago—"The physician who confines his attention to the body knows not the extent of his art. If he knows not how to soothe the irritation of a troubled and enfeebled mind, to calm the fretfulness of impatience, to rouse the courage of the timid, and even to quiet the compunction of an over-tender conscience, it will very much confine the efficacy of his prescriptions; and these he cannot do, without he gain the confidence, esteem, and even the love of his patients." I know of no one having incorporated in his makeup to a greater extent these qualities than had John Rogers.

The last three years of his life he was far from well. Following the death of his wife he was beset with evils of body as well as spirit. He had the misfortune to break his leg and was confined for some time. A year ago he had a rather severe gastric hemorrhage, which he feared might be of malignant origin, but which was later proven not to be. He refused to cease entirely seeing patients although he spent increasingly more time away from his office. But a year ago he made a world tour with his daughter. He had been about as usual and the news of his sudden death from heart failure came as a surprise on January 2, 1938.

In the year 1755 a little girl walked with her mother along a London street. Jostled to one side and being too tiny to see, she complained to her mother. The occasion was the passing of Samuel Johnson and some friends along the narrow sidewalks of Temple Bar. "Hush, child," said the mother to the little one, "a great man has passed by."

Upon ballot the following men were elected as candidates for membership in the Academy:

Dr. Albert Snell, Rochester, Associate Member.
Dr. Gilbert Thomas, Minneapolis, Active Member.

It was decided to have a non-scientific summer meeting at the country home of Dr. Archa Wilcox, and that Dr. Wilcox and a selected committee should decide just when the meeting is to be held.

The scientific program followed.

SPINA BIFIDA CYSTICA OF THE PELVIS

Diagnosis and Surgical Treatment

ALFRED W. ADSON, M.D.
Rochester, Minnesota

(See page 468, this issue)

Discussion

DR. F. C. RODDA, Minneapolis: We have had the following experience. Operation for the repair of spina bifida in infants two to four months of age, in which the hydrocephalus seemed to be quiescent, has been followed by a very sharp and rapid increase in the size of the head. On the other hand, where the operation has been delayed until the child has reached the age of ten or twelve months, we have not had this difficulty. It would appear as though the sac acted as a sort of safety valve in the early months of life. I should like to ask Dr. Adson if that is a factor in his selection of a nine months' age limit.

DR. F. R. WRIGHT, Minneapolis: What is the general outlook as to prognosis of these cases? Do they recover and make physiologically healthy individuals, or do they recover just sufficiently to be taken care of by somebody else?

DR. ARNOLD SCHWYZER, Saint Paul: I rise to discuss this paper because Dr. Wright has asked the question as to whether these patients are any good afterward. I saw, a few weeks ago, a man who came in with his mother who was sick. She said, "You know, I think some twenty-three or twenty-four years ago you operated on our baby. This fellow here is that baby." In this case there was a large meningocele in the upper cervical region, and this young man was a regular giant in size and strength. His neck showed a somewhat irregular scar. The worst forms are the myeloceles where the whole medullary canal is open. Inside of the edge of skin there is the zona serosa and, in the center a granular looking surface, the medullary tissue. In the median line above and below, one can see a little dimple which represents the entrance to the medullary canal. I have operated on two such cases, but there is not great opportunity to do much for the condition. Both babies lived, but the paralysis of the lower extremities persisted. The cord lies open in the center. For a couple of days before operation we try to get the area clean by using saline irrigations and a salicylic acid solution. Then the myelocele is closed. Both of these patients were paralyzed and stayed paralyzed, so far as we know, except that one of them, the child of a doctor, has shown considerable im-

provement in the feet. They are not entirely paralyzed.

(Note: Inquiry after the meeting from the father of this child with myelocele and an open central medullary canal reveals that the child can move the thighs and stretch and bend the knees. The feet are being treated orthopedically, and there is a grave sensory deficiency.)

Of tumors of the anterior area of the sacrum, Dr. Adson mentioned chordomas and teratomas. The first surgical case I had in my practice was a man sent to me by another doctor. The lesion was congenital. At birth it had apparently fluctuated, was incised by the attending physician, and never healed. The man was a poor farmer's helper and had always slept in the hay out in the barn because there was such a terrific odor about him. He was 18 years old when he came to see me. His was a presacral teratoma, between the sacrum and rectum.

A case of chordoma which I had in this area was brought before this Academy some time ago. [Meeting of October 7, 1936. Published in MINNESOTA MEDICINE, 20:15, (January) 1937.]

I should like to ask Dr. Adson if the sac in his case was injected with an obliterating fluid like alcohol.

DR. F. E. B. FOLEY, Saint Paul: This is one of the most interesting papers I have heard presented before the Academy. Dr. Adson has presented the subject of meningocele in a way most instructive and helpful to those concerned with the clinical surgical problem. My own interest is more in the physiology and pathologic physiology of the condition. This interest carries over from experimental work on the physiology of the cerebrospinal fluid done in association with Dr. Harvey Cushing in 1920. I would like to ask some questions bearing on this phase of the matter.

Is not the hydrocephalus developed following removal of a large meningocele always of the communicating type? Obstructive hydrocephalus due to occlusion of the aqueduct or the foramina entering the subarachnoid space should exist independently of the meningocele and not be influenced by its removal. Does occurrence of a communicating hydrocephalus in response to removing a meningocele indicate that the lining of the cyst had been a pathway for absorption of fluid and had protected the individual from hydrocephalus? Is removal of this absorbing surface directly responsible for the hydrocephalus?

In commenting on hydrocephalus secondary to removal of a meningocele, Dr. Adson mentioned the theory of Morgagni. This theory presupposes that the choroid plexus behaves as a secreting gland and that the cerebrospinal fluid is a true secretion. The experiments which I devised and carried out in Dr. Cushing's laboratory disprove this. They showed conclusively that the choroid plexus is not a secreting gland and that the fluid is a product of purely physical factors—filtration, osmosis and diffusion. A mixture of solutions of iron ammonium citrate and potassium ferrocyanid was supplied to the subarachnoid space from a manometer. The animal was then given a hypertonic salt solution intravenously. Incident to the increased absorption of cerebrospinal fluid and fall of fluid pressure thus induced, a considerable volume of the citrate-ferrocyanid mixture was displaced from the manometer into the subarachnoid space. On perfusing the animal with formalin containing hydrochloric acid, insoluble granules of Prussian Blue were precipitated from the mixture at the sites to which it had reached. It was found that a reversal of flow in the aqueduct had occurred and that fluid now moved from the subarachnoid space up the aqueduct into the ventricles. There was a heavy deposit of Prussian Blue over the ependymal cells of the choroid, between these cells and in the choroidal vessels. The experiment proved that the increased osmotic value of the blood had reversed the flow of fluid through the choroid plexus which now ceased production of fluid and became a pathway of fluid absorption. Some years later these experiments were repeated and verified by Freemont-Smith. They show conclusively that the fluid is a product of purely physical factors and is not a true secretion. May it not be possible to put this fact to good purpose in the surgical treatment of meningocele?

DR. ADSON, in closing: In reply to Dr. Rodda's question as to whether or not a balance develops between the secretion and absorption of cerebrospinal fluid in communicating hydrocephalus, I wish to say that it is my opinion that a balance does develop when there is not too large a difference between the factors involved; that is, when the absorption mechanism is capable of absorbing most of the fluid secreted.

In reply to Dr. Wright's questions as to whether or not surgery is worth while, I wish to emphasize the point that I made in the paper. This was that the surgical treatment of spina bifida cystica is not indicated in the presence of an extensive hydrocephalus, nor is it indicated in the presence of a syringomyelocele when extensive paralysis is manifest. In the remaining group of cases it is definitely indicated and worth while.

I doubt if surgery is of material value in the treatment of spina bifida occulta. Occasionally, adhesions may be relieved and a fatty tumor found which, when removed, may give some relief; more often than not, however, nothing can be done from the surgical point of view. The symptoms most usually are the result of a myelodysplasia.

In reply to Dr. Foley's question as to whether or not the choroid plexus is a secreting gland, I wish to state that I am familiar with his experimental work and agree with him that the cerebrospinal fluid is not a secretory product of the choroid plexus but is a transudate as a result of various physical phenomena. However, the point that I wished to make was that the cerebrospinal fluid was eliminated by the choroid plexus into the lateral ventricles and had to find its way into the subarachnoid spaces. Otherwise a hydrocephalus would result.

CASE REPORT

FREDERIC E. B. FOLEY, M.D., and
JOHN E. DEES, M.D. (by invitation)
Saint Paul

The case to be reported is that of a married woman, aged fifty-five (Ancker Hospital record No. A6941). The patient had been treated for three years in the cardiac clinic of the Ancker Hospital for auricular fibrillation. She was admitted to the hospital on January 8, 1938, complaining of severe pain in the left flank and back for the preceding thirty-six hours. The onset had been sudden. The pain was severe and constant, being localized in the left costo-vertebral region and flank. There was no downward radiation. On three occasions since the onset of the attack she had noted gross blood in the urine. There had been no other urinary symptoms, no chills or vomiting.

On admission significant findings on physical examination were a temperature of 100° F, marked obesity and extreme tenderness to palpation over the left flank and costo-vertebral region. No mass was palpable. There was a rapid auricular fibrillation with an apical heart rate of 160 per minute, the blood pressure being 120/90. Except for a few râles in the bases of the lungs, there were no signs of decompensation.

Laboratory examinations showed the hemoglobin to be 78 per cent, leukocyte count 25,800, sedimentation rate 55 mm./hour, urea nitrogen 28 mmg. per cent.

The urine showed an occasional leukocyte and granular cast, no erythrocytes, 1-albumin, no sugar and it was sterile on culture.

On January 10 x-ray of the abdomen was negative for a rather large left renal shadow. Intravenous urography showed a well functioning, apparently normal right kidney with no excretion of dye from the left kidney in thirty minutes. Cystoscopy was then carried out. The bladder was negative. A No. 6 catheter was easily passed to the left kidney. There was no drainage of urine from the catheter but irrigation of the left renal pelvis with normal saline solution reproduced the patient's pain. The catheter was left in place for five hours at the end of which time it was removed as there had been no drainage of urine through it. Retrograde pyelography was not carried out at this time as the patient's temperature was 102° F.

The temperature subsided slowly from a maximum of 102 on January 10, reaching normal on January 18, the tenth day of hospitalization. Spontaneous pain in the kidney disappeared by the fifth day of hospitalization and tenderness to pressure by the seventh day.

On January 18 the left ureter was again catheterized. There was no drainage of urine nor could any be aspirated from the renal pelvis. The ureteral catheter was removed and a bulb catheter wedged into the left ureteral orifice. A pyelo-ureterogram was made after the injection of 11 c.c. of 20 per cent hippuran solution, which amount reproduced the patient's pain. The resulting picture showed an essentially normal, unobstructed ureter and kidney pelvis. A diagnosis of massive embolism of the left renal artery was made at this time.

Two days later cystoscopy was again carried out. Indigo-carmine appeared in good concentration in three minutes from the right ureter but none during ten minutes observation was seen to come from the left side. A No. 14 bulb catheter was wedged into the left intramural ureter and left in place for thirty-five minutes during which time there was no drainage of dye or urine through it.

The patient left the hospital on January 23. At this time there were no symptoms referable to the urinary tract. The urine contained many erythrocytes and leukocytes. The tachycardia had disappeared under digitalis.

The patient has been re-admitted for study on three occasions since discharge, two, three and four and one-half months respectively after the onset of her illness. Intravenous urography and cystoscopy after the injection of indigo-carmine intravenously as well as ureteral catheterizations have consistently showed an absence of excretion of dye or urine by the left kidney. Bulb pyelo-ureterograms have shown a progressive decrease in the capacity of the left renal pelvis and ureter from 11 c.c. on January 18 to 4 c.c. on May 16, The size of the pelvis and renal shadow have decreased markedly, the general configuration of the pelvis being preserved in miniature. There have been no urinary symptoms or evidence of other embolic episodes.

Cursory review of the literature in connection with this case report discloses publications on the subject beginning in 1856 when Traube first reported the condition. Schultz, five years previously, had ligated the artery in an experimental animal and observed atrophy of the kidney. Blessig, in 1859, in a similar experiment concluded that the renal artery was necessary, not only for the function, but for the nourishment of the kidney. He found that its ligation was followed in the first few days by venous engorgement and that this might cause actual hemorrhage if thrombosis of the vein also occurred. This latter observation is difficult or impossible to explain reasonably. With total occlusion of the artery, hematuria should be expected only in the presence of a patent vein. In such event venous pressure in the cava being higher than in the venules of the kidney, there should be engorgement of the kidney with diapedesis and blood in the urine the result. Why bleeding should occur in the presence of occlusion of both artery and vein is hard to understand and would seem to indicate fault in the experiment.

Buchwalden and Littin, in 1876, ligated the renal vein and found initial enlargement of the kidney with shrinkage of the kidney beginning by the sixth day and finally with establishment of collateral venous circulation through capsular renal vessels and lumbar veins with some preservation of function. This was corroborated in 1913 by Morrell and associates. These investigators found that following ligation of the artery rapid necrosis, atrophy and death of the kidney invariably resulted.

Westerborn, in 1937, reported careful observations and operation in a case very similar to the one here described. He also made some animal experiments. He found that one and one-third hours is the longest period of arterial occlusion that will be followed by total recovery of the kidney. He reports that occlusion of an hour and a half invariably results in impaired function. He concludes that embolectomy if done at all should be done in the first hour and certainly not later than two hours.

Eisendrath, in 1934, reviewed the reported cases of both arterial and venous occlusion—some forty cases in number, and concluded that of them only twenty-five were acceptable examples of the condition.

The numerous publications do not sharply distinguish between total occlusion of the main renal artery, multiple occlusions of its smaller branches and venous occlusion. For this reason it is difficult to know the exact character of the vascular occlusion or combination of occlusions that was responsible for the clinical findings in the reported cases.

Consideration of the reports of massive renal infarct makes it appear that the characteristic onset is one of sudden severe pain, usually localized to the kidney region although at times being epigastric. The pain is without remission or radiation and usually of such severity as not to be relieved by morphine. In the majority of cases gross hematuria occurrs. Apparently it may be present with primary arterial as well as primary venous occlusion.

The reports are agreed that there is exquisite tenderness of the affected kidney with or without spasm of the overlying muscles. Albuminuria is the most frequent laboratory finding occurring in roughly 60 per

cent of the cases referred to. It tends to decrease or disappear by the sixth day.

The papers to which we have had access disclose only three cases closely analogous to the one here reported—that is embolism or sudden arterial thrombosis with complete occlusion of the whole renal artery. None of these reported cases contains detailed information concerning frequently repeated observations following the event though in two cases subsequent cystoscopy and pyelography are reported with findings similar to ours. The case we have described appears to be the most complete and detailed observation of the condition as clinically encountered.

The meeting adjourned.

Meeting of May 11, 1938

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, May 11, 1938. Dinner was served at 7 o'clock and the meeting was called to order at 8:10 by the president, Dr. R. T. LaVake.

There were forty-nine members and three guests present.

Minutes of the April meeting were read and approved.

The scientific program followed.

NON-TUBERCULOUS SPONTANEOUS PNEUMOTHORAX

E. V. Kenefick, M.D.
Saint Paul

Abstract

Spontaneous pneumothorax may be caused by tuberculosis, lung abscess, bronchiectasis, emphysema, empyema, carcinoma of the lung, lung cyst, and silicosis. Fracture of the ribs and puncture wounds of the chest were responsible for the first reported cases. Tuberculosis is the most common cause and is present in 70 to 80 per cent of all cases. Another small group without obvious cause are classed together because of a common symptomatology and course. Benign pneumothorax, idiopathic pneumothorax, and non-tuberculous pneumothorax are the various terms applied to this condition. It is this group I wish to discuss.

The various types of spontaneous pneumothorax encountered may be classified according to the degree to which the lung is collapsed. The collapse may be partial or complete, with or without mediastinal displacement. If the pressure continues to increase a tension, pneumothorax may develop. Recurrent attacks are common. Simultaneous bilateral pneumothorax may occur and is much more serious than the unilateral form. Hemopneumothorax is also a serious type, due to the presence of large amounts of blood in the pleural space.

The close relationship of pulmonary tuberculosis to spontaneous pneumothorax has been an accepted dictum for many years. Several reasons may be advanced to prove this is not true in all cases. Spontaneous pneumothorax occurs usually in the late stages of tuberculosis. If it does occur due to the rupture of a solitary focus in the pleura, a pleural effusion invariably results. In spontaneous pneumothorax, pleural effusion is not present and follow-up studies do not reveal the presence of pulmonary tuberculosis. Kjaergard has studied fifty-one cases, some of which were followed for as long as eighteen years, and found only one patient had developed active tuberculosis. Morriss studied twenty-six cases over a period of from three to eleven years, and found no tuberculosis. The more general employment of the tuberculin test and routine x-ray examination of the chest has demonstrated that an antecedent tuberculous infection is not necessary in the development of spontaneous pneumothorax.

The condition occurs most frequently in young males between twenty and thirty years of age. Physical effort or strain does not play any part. Ranking, Pitt, Hayoshii and Fisher have reported finding vesicles at the apices of the lung surrounded by areas of emphysema or retracted areas due to scar tissue. No evidence of tuberculosis was present in any of their autopsied cases, six in number. Fisher and Hayoshii ascribe the pneumothorax to the rupture of a vesicle, usually situated at the apex, which is surrounded by either scar tissue or emphysematous tissue, closed by a valve at the base. The valves develop from inflammatory processes with resulting scar tissue formation, producing a constriction of the lung tissue or bronchiole.

Case histories of seven patients with spontaneous pneumothorax were presented together with the results of tuberculin tests and x-ray studies of the chest. These studies failed to reveal evidences of tuberculosis in any of the patients. The follow-up period was five years in all cases. Mantoux tests were negative except for one patient with a plus reaction three years after the attack of pneumothorax. Fever was not present and there was no evidence of pleural effusion in any case. All patients completely recovered in from two to six weeks with simple bed rest and symptomatic care.

CONCLUSIONS

1. Non-tuberculous spontaneous pneumothorax occurs in healthy young adults without demonstrable cause and runs a benign afebrile course with complete recovery in a few weeks.

2. The condition is caused by the rupture of a pleural valve vesicle either emphysematous or scar tissue in nature.

3. Tuberculosis is not a factor in this type of spontaneous pneumothorax.

4. Roentgenology and tuberculin tests are extremely valuable aids in the diagnosis and treatment of this condition.

Discussion

Dr. E. V. Goltz, Saint Paul: I recently had an opportunity of observing just such a case as Dr. Kene-

fick reports, and his paper has been very interesting to me because of this recent experience, for his cases simulate mine very closely. It seems to me the interesting observation here is the etiologic factor. While it is true that some claim 80 per cent of their spontaneous pneumothorax cases to be tuberculous, I think this probably holds true more in hospitals and sanatoria. In private practice I think one can not claim so high a percentage. The problem is to prove that these young people who have these benign spontaneous pneumothoraces are not tuberculous. Dr. Kenefick has shown very definitely that his group is not tuberculous; and, if this can be proven, it is certainly very much worth while in the prognosis.

DR. S. MARX WHITE, Minneapolis: One patient of mine at the age of 32 first had a spontaneous pneumothorax in 1928. He had two subsequent recurrences, each after about a year's interval, and two of the three attacks were brought on apparently by riding horseback. At the time of the first attack minor symptoms of a chronic type of arthritis were present, and subsequently a moderately severe extensive Strümpel-Marie type of spinal arthritis has developed. The Mantoux reactions have all been negative, and repeated x-ray films, taken at the time and subsequently, to the present time have been negative to tuberculosis.

A fact of interest is that a paternal uncle had very marked and extensive cystic disease of the lung, found at autopsy. In the x-ray films of this patient, however, we have not been able on careful search to find anything indicative of cystic disease of the lung, even though this was searched for at the time of the attacks.

DR. MAX HOFFMAN, Saint Paul: In the last two years I have seen three cases of spontaneous pneumothorax. None of them showed much air in the chest and in all cases the symptoms were minimal. There was some pain in the chest underneath the clavicle on the affected side, but there was much less air than in the cases Dr. Kenefick reported tonight. That would make one feel that spontaneous pneumothorax of mild degree is fairly common. The pain may last for a day or two and the condition may heal quite quickly. Last year I read an article in which the author had injected 25 per cent glucose into the thoracic cavity for this condition. He felt it was very effective in preventing a return of spontaneous pneumothorax. I have not had a chance to use this treatment.

DR. C. M. CARLAW, Minneapolis: I was very glad to hear Dr. Kenefick's paper. It has taken me back many years. About the year 1890 at the Montreal General Hospital, Dr. George Ross, then Professor of Clinical Medicine at McGill, in one of his clinical lectures to the class of which I was a member, explained and demonstrated to us the use of the "Coin Test" in the diagnosis of pneumothorax. The second year after I was graduated I came to Minneapolis. One night shortly after, I was called to see a young man who was suffering with severe chest pain. I could not at first arrive at any diagnosis. Suddenly, however, I remembered Dr. Ross' lecture on the "Coin Test," tried it, and there was a pneumothorax as plain as the nose on his face. I made some inquiry into his family history and found that his mother had died of consumption, also an uncle on his mother's side. The patient looked as though he might have it too. That man got well after about two weeks' rest in bed. He is now an officer in one of our Minneapolis banks, has reared two fine healthy children, and has just been blessed with a fine big grandson; and he (grandpa) still does not show any signs of tuberculosis.

DR. KENEFICK, in closing: I want to thank the members for their kind discussions. There are a few points I wish to mention. Dr. White mentioned the possibility of lung cysts as a cause of pneumothorax. A number of these cases have been reported. I know of two cases where x-rays taken at the time of the pneumothorax showed a cyst which had ruptured and remained after the lung collapse. I believe in Kjaergard's monograph there is a nice picture of a lung cyst with a pneumothorax.

Dr. Goltz mentioned the differences of opinion as to the incidence of tuberculosis in these cases. I think that is perfectly true. Men who are doing most of their work with tuberculosis in institutions have an idea that these are all tuberculous because they are dealing mostly with tuberculosis. On the other hand, in private practice one probably sees many more cases of the simple type.

Dr. Hoffman mentioned the injection of glucose. This is recommended in cases of hemopneumothorax to cause the blood to clot. The injection of sterile water or glucose, or almost any foreign protein, seemingly causes coagulation and stops the hemorrhage.

These cases are mainly of interest because of the prognosis. If one takes for granted that these patients are tuberculous and keeps them in bed for a long time with the stigma of tuberculosis, naturally they lose much time. With the use of the Montoux test, a large percentage of these cases are proven not to be tuberculosis.

The meeting adjourned.

A. G. SCHULZE, M.D., *Secretary.*

ACUTE LYMPHATIC LEUKEMIA WITH REMISSION (CASE REPORT)

(Continued from Page 501)

area of consolidation or dulness in the left base had disappeared entirely by April 9, leaving normal breath sounds. The child seemed to gain a little in strength, her color improved, she was brighter, she responded better and on April 6 began to eat rather generous amounts of solid food, the first solid food she had taken for two weeks. Her pulse became somewhat slower, changing from 140 and 150 to 130. The temperature, however, was never less than 100 by axilla and it would rise very easily to 104 by axilla. On April 9, her white count was 64,000, a reduction of almost 100,000 cells per cm. over a period of ten days. The blood smear continued to show the typical picture of acute lymphatic leukemia. From March 30 to April 10 eight injections of liver extract were given. April 9, a pink generalized maculopapular eruption was first observed. This persisted until her demise five days later.

April 10, the patient began definitely to lose ground. Bleeding from nose, mouth, and rectum began. Breathing was rapid and shallow. The pasty pallor returned and the responses were very poor. There were a few scattered leukemic infiltrations in the skin still remaining, but neither did these increase nor did any areas of dulness return to the chest. Indeed, the skin nodules from the time they began to subside until exitus continued to do so although much more slowly the last few days. April 11, the child had a few mild convulsions. General appearance was one of pallor, bloating, extreme toxicity. Exitus occurred April 14.

BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

HANDBOOK OF ORTHOPÆDIC SURGERY. Alfred Rives Shands, Jr., M.D., and Richard Raney, M.D. 593 pages. Illus. $5.00. St. Louis: Mosby, 1937.

I find this an adequate outline covering the subject adapted to the needs particularly of students. The drawings illustrate deformities and bone pathology in many cases better than the average photographs of radiographs. The bibliography is comprehensive and well organized and the index accurate.

S. W. SHIMONEK

MODERN TREATMENT AND FORMULARY. Edw. A. Mullen, M.D. 707 pp., $5.00. Philadelphia: F. A. Davis Co., 1936.

Anyone interested in the essentials of treatment including numerous authenticated prescriptions, and diet lists, tables of differential diagnosis, and other valuable data and tables, will find these compactly embodied in this well-organized and readable volume. It should prove a valuable handbook, particularly for the general practitioner.

THOMAS MYERS, M.D.

MANAGEMENT OF THE SICK INFANT AND CHILD. By Langley Porter and Wm. E. Carter. 5th Ed., 874 pp. $10.00. St. Louis: C. V. Mosby Co., 1938.

This excellent discussion has been justly popular, having gone through six printings in sixteen years. Despite its title, the work is not limited solely to a discussion of treatment, but also devotes ample space to etiology, symptomatology, and description of the various manifestations of diseases. Over two hundred pages are devoted to a well-illustrated discussion of methods of treatment, briefly and simply outlined. Diets in disease and health are competently described, and appropriate recipes and tables are included. A valuable chapter on drugs with appropriate prescriptions and dosage in various conditions completes this volume. The typography and excellent paper used make the book easily readable. Certainly this is an indispensable addition to the library of anyone treating children.

THOMAS MYERS, M.D.

INJECTION TREATMENT OF VARICOSE VEINS AND HEMORRHOIDS. H. O. McPheeters, M.D., and James Kerr Anderson, M.D. 315 pages. Illus. $4.50. Philadelphia: F. A. Davis Co., 1938.

The section on injection of varicose veins by H. O. McPheeters is a very comprehensive treatise on the anatomy, embryology, etiology and diagnosis of this condition. There is an excellent and clear description of the physiology involved and of the Trendelenburg test. The chapters on treatment are extremely clear and easily followed. The few hours necessary to read this is time well spent.

The section on the injection treatment of hemorrhoids by James Kerr Anderson is the clearest, most concise, and most thorough description of this subject that one can find. The chapters are written with clearness and brevity that is welcome. Obscure points are brought forth in easily understood terms and there is no doubt in the reader's mind as to the author's meaning. It is well worth reading by everyone who contemplates injecting or is injecting hemorrhoids. The reading time is a little over an hour.

WALLACE P. RITCHIE

IN MEMORIAM

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whom I have ever known. Intolerant of anything but the highest type of professional conduct, he was never influenced by anything commercial in his professional life. He maintained in his surgical diagnosis and technic a perfection that few attain.

The life and work of the medical man are brief and often too soon forgotten but the great principles of ethical conduct to which he was committed are age-old and permanent. I like to believe that the memory of Dr. Wiese will live for all time in these great principles.

CLAUDE C. KENNEDY, M.D.

L. G. Wilberton

1853-1938

Dr. L. G. Wilberton, a practicing physician at Winona for fifty-eight years, died at his home there on May 29. Dr. Wilberton was reared in New York State, and was a graduate of Hahnemann College at Philadelphia, in the class of 1880, coming to Winona directly after his graduation. Surviving him are his wife and two children, Mrs. Ernest E. Shepard of Winona and George L. Wilberton of Seattle, Wash. Dr. Wilberton was a member of the American Institute of Homeopathy and for a time was vice president of the Minnesota State Homeopathic association. He was the oldest homeopathic physician in Minnesota.